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EPILEPSY

AND OTHER

CHRONIC CONVULSIVE DISEASES:

THEIR CAUSES, SYMPTOMS, AND TREATMENT.

BY

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ROYAL SOCIETY OF SCIENCE OF UPSALA.

SECOND EDITION.

LONDON:
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THE MEMORY OF

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PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS
PHYSICIAN TO HER MAJESTY'S HOUSEHOLD
CONSULTING PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL

WHOSE EXAMPLE STIMULATED

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40628

PREFACE TO THE SECOND EDITION.

THE original character of the book has been maintained, but every sentence has been revised, numerous additions have been made, and many parts have been re-written. The statistical basis, from which the more general facts of the disease have been ascertained, is increased from 1450 to 3000 cases, and the number of cases from which other features have been ascertained is augmented in similar proportion. To the time and labour involved in such comparative observation is due the delay in the appearance of this edition.

I am much indebted to Dr. James Collier for the preparation of the Index and a final perusal of the proofs. Mr. Victor Horsley has been kind enough to consider carefully the final chapter on Surgical Measures.

QUEEN ANNE STREET, LONDON;
May, 1901.

PREFACE TO THE FIRST EDITION.

THE object of the following work is to describe and illustrate the clinical history of Epilepsy and other allied convulsive diseases by the help of the material furnished by a series of cases which have been under my care, chiefly at the National Hospital for the Paralysed and Epileptic. Some of the facts of these cases, and the conclusions which they suggest, were described in the Gulstonian Lectures delivered before the Royal College of Physicians of London in February, 1880. Those facts and conclusions are here reproduced, in their proper place, in the systematic account of these diseases. The lectures, as published in the medical journals, do not, however, occupy more than a fourth part of the present volume.

Many of the conclusions given in the following pages are the same as those which other workers have reached; others are different. It has not been practicable to refer in every instance to the numerous writers who have expressed opinions on the several debated points. Whatever value is possessed by the inferences here presented is due to their being drawn from an extensive series of facts in an original investigation. I have, however, referred to the statements and opinions of others whenever justice or the importance of the facts seemed to render such reference desirable.

QUEEN ANNE STREET;
September, 1881.

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INTRODUCTION.

THE disease which, following Hippocrates, we call "Epilepsy," was known to our English forefathers as the "Falling Evil," after the Roman *morbus caducus*.[*] The latter name presents the most common feature of the disease, the former only a pseudo-mythical pathology. The characteristic of the malady is the recurrence of sudden brief disturbance of some functions of the brain, varying in degree, extent, and character, but generally attended with an arrest of consciousness sufficient at least to interrupt the control of the muscles necessary for the maintenance of the erect posture. The sufferer therefore falls, and hence the designation "falling-evil," replaced by "falling-sickness" when the word "sickness" came to be a general name for "disease."[†]

---

[*] 'Ancren Ruele' (circa 1200, edited by Morton, Camden Society, 1853), p. 176. "Fallinde veul ich cleopie lecomes sicnesse" ('vuel' = yvel). The whole sentence, as translated by Morton, is worth reproducing: ". . . . Another property, which is very good for an anchoress, although it is hated; that is the falling sickness. For it is very necessary for an anchoress of holy and highly pious life to have the falling sickness. I do not mean the sickness which is commonly so called, but that which I call falling sickness is an infirmity of the body, or temptation of carnal frailty, by which she seems to herself to fall down from her holy and exalted piety. She would otherwise grow presumptuous, etc." This passage shows how familiar the word was 150 years after the Norman conquest, and makes it probable that the name had been in use for centuries.

[†] The Germans also termed it "Fallsucht." Although "morbus caducus" was applied to it by the Romans, they more commonly called it "morbus comitialis," because the occurrence of a fit in a public assembly (comitia) was deemed so unpropitious that the meeting was broken up. By the Greeks of the time of Hippocrates it was commonly termed the "Sacred Disease," on

But there is usually far more than the fall. The disturbance commonly involves the sudden release of nerve energy in the cerebral centres. The motor centres are especially prone to be involved, and the result is contraction of the muscles, sometimes slight but often intense, giving rise to what is called a "convulsion." The whole frame is fixed by "tonic" spasm in some strained posture, unlike that which is ever seen in normal states. The chest becomes rigid and the respiratory movements are arrested; the face becomes congested and livid. When life seems almost at an end, the muscular spasm presents remissions so as to become "clonic." The remissions are at first slight and frequent, but gradually become deeper and less frequent, so as to assume the character of jerks, and the relaxation between them becomes greater until they amount to sudden shock-like contractions. These effect some renewal of air in the lungs and relieve the state of asphyxia. Frothy saliva, often blood-stained, is extruded from the lips, and when the last jerk has occurred the sufferer is left prostrate and unconscious. To the observers of such an attack, when all such phenomena were ascribed to unseen but personal influences, it seemed that the attack must be the work of some malignant agent, and so it was called "epilepsy," a "falling on," and a trace of the same thought seems to underlie the familiar term "seizure," and is not quite absent from "attack."

All our present knowledge suggests that the primary disturbance of function which underlies the epileptic fit occurs in the cerebral cortex. It may begin, not in the motor centres, but in those that receive impressions from the periphery, in the sensory centres. The first symptom is, then, the effect on consciousness of a "sensation," referred to some part of the

---

account of the assumed supernatural cause of the attack. The term "comitial disease" obtained some use in English (1562, Bulleyn; 1598, Sylvester; 1627, Burton). The first form of "epilepsy" was "epilency" (1398, Trevisa). In 1607 Topsall recommended against the "falling sickness" the "gall of a ferret." In 1652 ('Women's Universe') we have a sarcastic allusion, probably to hysteria, which may even then have given rise to the diagnostic difficulty it still presents to doctors who—

> "Could never cure her falling-ill,
> Which takes her when she pleases."

body or to one or more of the special senses. As the process spreads in the brain, consciousness is commonly lost, and muscular spasm and convulsion are often superadded. The latter may be absent in slight attacks, to which the term "sensation" is then popularly applied. A slight disturbance of the motor centres may give rise only to brief giddiness, or motor spasm may be local and limited, or may consist only of slight general rigidity. Other slight attacks may consist of brief loss of consciousness alone, and sometimes only of its momentary impairment. The slight attacks, without conspicuous muscular spasm, are commonly termed "minor epilepsy" or "petit mal"; those with marked convulsions, "major epilepsy" or "grand mal."

When an attack begins with sufficient deliberation for the commencing process to influence consciousness, the effect is termed the "warning" or "aura" of the fit. The origin of the latter term will be afterwards explained. These warning sensations are extremely various, pronounced, and elaborate, or dim, and sometimes absent. But they constitute an important element in the symptomatology of epilepsy.

Although a fall is a more general feature than is a convulsion, characteristic symptoms may occur without a fall. The use of the term "epilepsy" has the adequate justification that it has now lost its etymological significance, and is sufficiently unmeaning to involve no error and to embrace all the symptoms and forms of the disease, just as the use of "hysteria" is justified by its convenience, although we no longer believe that the symptoms are produced by an errant womb, which wanders about the body.

The features presented by the epileptic attack are thus so various that it is scarcely possible to frame an adequate clinical definition of the disease. But the symptoms, although they vary extremely, possess always the characteristics of suddenness in onset and brevity in duration. They sometimes seem to last longer, but this is by reason of a secondary and subsequent disturbance of the brain; and those of a truly epileptic nature endure only for a few minutes, and often only for a few seconds.

Another important and indeed essential element in the disease is the tendency to the recurrence of the attacks. A single convulsion, however characteristic, does not constitute epilepsy. As a disease, it consists in the repetition of attacks, which depends on the fact that every functional state of the brain, normal or abnormal, leaves behind it a condition in which the same functional state occurs with greater readiness. The effect is the greater the more often the functional action has occurred. The tendency to the recurrence of attacks of epilepsy of every form is increased by each one. Every fit, slight or severe, is in some degree the effect of those that have preceded it, the cause of those that follow it. This residual disposition to repetition of the same activity is the physical basis of memory, of muscular training, of all cerebral education, and it is the basis of the morbid education of the brain which underlies epilepsy. The recognition of this is essential for an adequate comprehension of the causation of epilepsy, and also for the principles of its successful treatment.

If we can perceive the conditions that underlie normal action in the nerve-centres, the phenomena of epilepsy will become in some degree less mysterious. In health, energy is liberated in instant response to a definite stimulus. Such capacity for instant activity involves a delicate equipoise of the processes for the liberation of nerve force and for its restraint or control. The balance must depend on the processes of nutrition in the nerve structures, for the liberation of energy depends on the occurrence of chemical processes under the influence of life—processes which must be ever on the verge of disturbance. But the conditions of nutrition must involve constant tranquil molecular changes, and, as a consequence, some degree of equally tranquil functional activity. The store of latent energy must be maintained by nutritional processes, which involve a constant overflow of energy, as from a vessel filled to the brim and delicately poised, into which water is gently flowing, but the equilibrium of which is easily disturbed, with a resulting energetic discharge.

We can discern evidence of such continuous "overflow' so many parts of the nervous system as to justify viction that it obtains in all. We see it in the activity of the spinal cord which maintains muscular tone, and this must be accompanied by a similar continuous activity of the cerebral centres, of which there is indeed abundant evidence. Thus a condition of stability is maintained, and yet readiness for the energetic liberation of nerve force when a stimulus is received. In epilepsy, however, the equilibrium is apparently unstable, and a sudden release of energy occurs apart from a normal stimulus, in what we term "spontaneous discharge." The tendency to it must depend on some slight difference in the molecular constitution of the nerve structure, inherited or acquired. When we consider the energetic release of force in the motor centres, which the mere sight of an alarming object will instantly induce in any animal, it is intelligible that a trifling change in elementary constitution may involve the liability to apparently spontaneous action.

The activity thus suddenly developed in epilepsy is moreover abnormal in form. The combination of cerebral centres involved differs from that which is active in normal conditions, and the form in which the nerve energy is released is also unlike that which occurs in health. But the difference varies in degree; the more intense the discharge, the greater is its difference from that of health. It is greater also in some centres than in others. The most conspicuous deviation is seen in the discharge in the motor structures, manifested by the spasmodic muscular contractions, tonic and clonic, which are, as a rule, unlike any that are seen in health. When the discharge begins in a sensory centre, the process may differ but little from normal action, judging from its effect on consciousness. It produces a sensation which often resembles that which might be experienced in health from an adequate stimulus.

It is important to note, at the outset, these features of the epileptic discharge; an abnormal form of activity is associated with undue readiness to act. Both must be ascribed to nutritional changes, which permit the molecules to break up too readily and to escape in a more random manner than in health. It may be a congenital imperfection of nutrition, or induced by abnormal influences to which the sufferer has been exposed; it may be widespread in the brain or limited in extent; in the latter case it may be the result of a local cerebral lesion, stationary or active. The convulsions from active disease are not regarded as coming into the category of epilepsy, although notice must be taken of them on account of the light they throw on the features of some true epileptic attacks.

The lesion which is the cause of organic epilepsy is always in or near the cerebral cortex, and generally in or near the motor region. The attacks commence by spasm in that part of the periphery related to the cortical centre that is damaged—in the face, arm, or leg. The attacks differ from those common in idiopathic epilepsy by commencing with a sensation or with clonic spasm in the related part; the clonic spasm may spread and become general, changing to tonic spasm if it gets more intense. Consciousness is then usually lost. Slight attacks due to such an organic cause may be limited to spasm at the part in which the symptoms commence, and may be attended by no affection of consciousness.

In all cases, however, the morbid process of "discharge" promotes its recurrence, and does so in any part of the brain to which it may spread, whether it is the result of a general nutritional disposition or caused by local disease. The result of this residual disposition is to bring together the recurring convulsions that result from local disease, and those due to a general morbid tendency. The discharges set up by local disease spread in the brain, and wherever they extend they leave the same disposition to recurrence. The result is that such cases of "organic epilepsy" come to resemble "idiopathic epilepsy," and the longer they have continued the greater is the effect, so that the causal disease becomes subordinate to the general tendency induced. But the two classes present an important difference in that the acquired disease, resulting from an old local lesion, does not involve transmission to descendants, as does that which depends on a perversion of nutrition, congenital in the strictest sense of the word and often inherited.

Convulsive attacks, resembling those of epilepsy, occur in the toxæmic state induced by kidney disease, especially during pregnancy and its termination. These are commonly distinguished as "eclampsia," or "puerperal eclampsia." They cease if the patient survives, and are not related to true epilepsy. The term "eclampsia" is also sometimes applied to the convulsions which occur in young children at the time of the first dentition, and are due to retarded development of the brain. These have a more intimate connection with epilepsy, and may persist as such. This is also sometimes true of the convulsions which may be excited by reflex irritation, as of the intestinal tract. Persistence of attacks due to such causes is always facilitated by an inherited tendency to the disease, and this is also true of their occurrence. Such a tendency promotes the special effect of the exciting cause.

Another form of convulsive attack is often met with which is the result and expression of the morbid state of the brain, termed "hysteria." It is conspicuously different from the epileptic fit in its characters, and yet, in its more severe forms, presents intense muscular spasm. Hence it is not rare to have confusion between the two, different as they are in character and nature. Such attacks present all gradations of degree, but all present the characteristic feature that the spasmodic action is such as might be produced by the will, although they may transcend in degree voluntary movements. The movements are "co-ordinated," often quasi-purposive in character, and differ entirely in their aspect and character from the spasm of the epileptic fit. The slighter forms consist simply in the intense expression of emotion, in tears, laughter, clenching of the hands, without interference with consciousness. In more severe forms the co-ordinated spasm is most intense, and consists in arching of the trunk, quickly repeated movements of the limbs, lasting for a short time and recurring, the periods of spasm being separated by intervals of simpler emotional manifestation, often delirious in character. Such a serial alternation may last for an hour or more unless cut short by treatment. For the more severe attacks the designation "hysteroid" was proposed by the late Sir William Roberts, to escape the trivial and misleading associations of the term "hysterical," and the designation has sufficient practical convenience to warrant its adoption.

The features of all these seizures are described in the following pages. The chief subject of the work is true epilepsy, the varieties of character and course which the disease presents, its various relations, and its treatment.

CHAPTER I.

ETIOLOGY.

THE causes of epilepsy may be divided into those that are remote, and those that are immediate,—the predisposing and the exciting causes. The exciting cause of the disease is that of the first fit, because the malady is self-perpetuating. This first fit leaves behind it a tendency to recurrence, which is, indeed, often an increase in the primary disposition. For the most part the immediate cause is trifling, and the remote cause, the predisposition, is incomparably the more important of the two. Most of the exciting causes of the first fit, even when their influence is most direct, are quite inadequate, alone, to account for its occurrence. They are such as are daily encountered with impunity by normal individuals, and their special effect is due to the antecedent state. In a large number of cases, indeed, no exciting cause can be ascertained; the first attack is due solely to the gradually increasing tendency, or is immediately produced by some influence that is too slight to be either noticed or ascertained.

The malady is thus self-perpetuating; when one attack has occurred, whether as a result of an immediate excitant or not, others follow either without any immediate cause, or after some very trifling disturbance. This is due to a fact, already referred to, that every definite action of the nerve centres, in whatever form, leaves a disposition to the recurrence of similar functional activity. It seems that the renewal of the molecules, lost in the release of energy, occurs in the same combination but with some minute difference, facilitating a similar change and release of energy in similar form. The search for the causes of epilepsy must thus be chiefly an investigation into the conditions which precede the occurrence of the first fit. The excitant is like a spark which falls harmlessly on a heap of sand, but on gunpowder induces an explosion. The spark is the immediate cause; the real cause is the predisposition involved in the chemical constitution of the material on which it falls. When this is realised, the trivial character of many excitants ceases to be surprising. Nor should it be surprising that an exciting cause is often imperceptible. The disposition seems to increase at certain periods of life, as the nutritional constitution of the nerve tissue changes under the influence of development or of decay. With augmenting instability, equilibrium may fail under some definite disturbance, or only when its deficiency brings it within the influence of the slighter forces that act on every person every day.

PREDISPOSING CAUSES.

The relation of a disease to the general conditions under which each individual lives, shows how far they influence the occurrence of the malady.

SEX.—Females suffer from epilepsy rather more frequently than males. In the total number of cases analysed, 3000, females preponderate over males, the proportion being 13 to 12—females 52 per cent., males 48 per cent.; that is, to every 100 males there were 108 females. This is not due to accidental circumstances, for at the hospital in which a majority of these observations were made, in the cases which do not present convulsions, males preponderate.

In the first edition of this book the facts were ascertained from 1450 cases, and to them 1550 others are now added. The sexual percentage of each series and of the whole are these:

First Series. 1450 cases.	*Second Series.* 1550 cases.	*Total.* 3000 cases.
F., 53·4 per cent.	F., 50·7 per cent.	F., 52 per cent.
M., 46·6 „	M., 49·3 „	M., 48 „

It is reasonable to assume that the percentage of the total is very near the truth, and that thirteen females suffer to every twelve males.

In most statistics of epilepsy hitherto collected in this country males have preponderated. This is probably due to the inclusion of cases in which recurring convulsions were the consequence of an organic cerebral lesion. Such disease is usually due to injury or to syphilis, and both of these causes influence males more frequently than females. Most similar statistics were collected before we had learned to separate such cases from idiopathic epilepsy. Yet the proportional affection of the two sexes given above is nearly the same as that ascertained by Herpin.* The relation of sex to age will be presently considered.

HEREDITARY PREDISPOSITION.—There are few diseases in the production of which inheritance has more manifest influence,† and the traceable influence is always far less than that which exists. In examining inherited influence it is necessary to inquire—(1) How is its existence shown? (2) In what proportion of cases can it be traced?

It is well known that the "neuropathic tendency" does not always manifest itself in the same form, but it is not easy to discern the relation of its varieties. Many diseases of the nervous centres have no relation to that tendency of which epilepsy is a result. Some of the most grave are the result of morbid states of the blood-vessels, and are due to disease of organs, as the heart and kidneys, far removed from the nervous system, or are produced by a peculiar state of the blood. We cannot regard cerebral hæmorrhage, or necrotic softening of the brain from arterial occlusion, as primary diseases of the nervous system, although they occur within it, and are manifested by sudden disturbance of its function.‡ The chief morbid states (besides epilepsy itself) through which the same neuropathic tendency is manifested are insanity, and, to a much smaller degree, chorea, chronic hysteria, migraine, and some chronic forms of disease of the brain and of the spinal cord. Intemperance is probably also due, in many cases, to a neuropathic disposition, but is seldom to be trusted as its evidence.

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* 'Du Prognostic et du Traitement de l'Épilepsie,' Paris, 1852.

† "An epileptick son doth often come from an epileptic father."—Jeremy Taylor, 'Of Repentance,' p. 16.

‡ Herpin, who carefully investigated this question, found that the frequency of apoplexy among the relations of epileptics was positively smaller than in the population at large. Even if the conclusion is not perfectly accurate, it affords a useful emphasis on the statement in the text.

In every form of disease special influences tend to obscure or conceal the facts, due sometimes to the nature of the malady, sometimes to popular ideas which have led to reticence. The latter influence is especially effective in the middle and upper classes, in which a morbid family tendency may hinder marriage, and is also often regarded as involving a social stigma. The reticence that is most effective is that of a preceding generation, who have passed beyond the reach of questions. To this must be added the ignorance and the obtuseness of the poor, increased by unfamiliarity with the words employed. It is always needful to remember how small is the number of words they use or comprehend. A mother who had answered "No" to the question if any of her epileptic son's relations suffered from fits, presently re-entered the room with two other sons also epileptic,—brothers were of too near kin to be "relations."

Many other diseases, outside the nervous system, are often assumed to indicate a family tendency which aids in causing epilepsy. Some of them have this significance, but are so common, apart from epilepsy, that they cannot be admitted to have the weight ascribed to them. Others are neither so frequent in association with epilepsy nor of such a nature as to make the connection intelligible. A causal relation is often asserted on the strength of one or two striking instances, which wider observation fails to confirm. This is true of phthisis, and of other diseases of which ancestral or collateral trace is to be found, sometimes or often. They are occasionally conspicuous from the side of epilepsy; but when the relations of these diseases are studied, epilepsy is not to be discerned more distinctly than in the population at large. It is certain, therefore, that an erroneous opinion may readily be formed (see p. 9).

In all investigations into inherited tendencies the facts ascertained fall short of the truth on account of the conditions of the investigation,—preceding or associated disease in other members of the family cannot be ascertained in a considerable proportion of the cases in which it exists or has existed. The causes of this have been just mentioned.

In attempting to discern the influence of inherited tendency, notice can be taken of two diseases only. Epilepsy and insanity are interchangeable in families; they are certainly correlated; other "neuroses" are so widely related, or at least seem to be connected with so many other morbid states, as to make their significance uncertain. Even hysteria is too closely related to the normal nervous system of females to permit it to be of definite significance. Migraine is too closely connected with inherited gout, and so also is neuralgia, to permit weight to be assigned to them, and the relationship of epilepsy to gout is undecided. Dipsomania has more claim to a distinct connection, but is infrequent. By thus confining attention to the occurrence of epilepsy and insanity, the facts are more certain.

Of 2400 cases in which the point was carefully investigated, such inheritance was traced in 973, or 40 per cent.* But this, for the reason already given, is much below the truth. The series investigated for the first edition of this book showed 35 per cent. of inheritance (429 out of 1218 cases). These were almost all hospital cases. In the second series, seen since the first edition was published, the percentage is 46 (544 out of 1182). The cause for the increased proportion of inheritance in the second series is sufficiently evident and instructive. Almost all the cases of the first series were seen in hospital practice, and the poor often know little, and seldom know much, of the diseases of their relatives and ancestors. The second series includes a considerable number of cases seen in private. In the better classes there is far more knowledge, and seldom intentional concealment from a physician, of known facts. There is often, indeed, within a family, ignorance of hereditary disease, which is supposed to entail a stigma, and does involve apprehension. In consequence of this, one generation may be unaware of such disease in that which preceded it. The facts, therefore, fall short of the truth, but the effect of this influence is far less than that of ignorance among the lower classes.*

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* The proportion of cases in which evidence of inheritance is to be traced was found by Eccheverria ('On Epilepsy,' New York, 1870, p. 183) in 300 cases to be 28 per cent., by Reynolds ('Epilepsy,' &c., London, 1861, p. 124) from a much smaller number to be 31 per cent.

If the cases seen in private are taken separately they amount, in the two series, to 600, and heredity was traced in 47 per cent.

It is certain that even these facts are below the truth, and also that we are justified in assuming that the indications they afford are true of all classes. We may safely conclude that in at least 50 per cent. of all cases of epilepsy the malady is ultimately the result of neurotic inheritance. The proportion may be much larger, but its extent is not at present to be discerned.

Heredity and Sex.—Inheritance does not affect the two sexes equally, as is shown by the following figures:

	Males.	*Females.*
Without heredity .	735, or 61 per cent.	692, or 57 per cent.
With heredity . .	458, or 39 „	515, or 43 „
	1193	1207

Heredity thus appears to play a larger part in the causation of the disease in females than in males. Among those in which the disease was inherited, 973 cases, the females constituted 53 per cent., and the males only 47. It seems, therefore, that when there is an inherited taint the females of a family are more likely to suffer than the males. The same fact has been noted by other observers. In examining the mode of inheritance we obtain some light on the origin of this difference.

The inheritance is from the mother's side rather more frequently than from the father's, the difference amounting to 13 per cent., as shown in the following cases of 975 inherited cases in which the point was noted:

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* The following instance illustrates at once the concealment in the better classes, and the frankness with which a doctor is usually treated:—A lady brought her son aged four, who had had fits since a year old. She only knew that a half-cousin of her father's had had fits. But the lady's mother came also, and stayed behind to tell me that her husband, the boy's grandfather, and two of his sisters were epileptic. Of this the patient's mother knew nothing. In a few years the knowledge would have disappeared from the family. This instance gives weight to the opinion that, even in the better classes, ascertainable facts fall short of the truth.

Inheritance.	*Cases.*	*Percentage.*
Father's side	333	34·8
Mother's side	361	37·7
Both sides	65	6·7
Collaterals only	198	20·8
	957	100

The side from which the disease is inherited has another relation; it has a distinct influence on the occurrence of the affection in the two sexes. There is clearly a tendency for the transmission to be from father to son and from mother to daughter. In the cases in which the disease was inherited from the father's side, males were in excess of the females by about 13 per cent. (188 males to 145 females, equivalent to 56 per cent. males and 44 per cent. females), which is an excess of 9 per cent. above the average for the whole number (47 per cent. males). Whereas, in the cases in which the disease was inherited from the mother's side, females were in excess by about 17 per cent. (16 per cent. in the first series—98 to 71, but 18 per cent. in the second—100 to 67). The percentage of the males affected is thus 29 per cent. greater when the disease is inherited from the father's than when from the mother's side.

The affection of brothers or sisters must be regarded as an indication of congenital tendency, although we may be unable to trace antecedent instances. This feature is often met with in diseases that may be inherited. Many members of a family may present the disease, so far as can be learned, *de novo.* It is frequent in phthisis, and is a very important although mysterious fact. In the cases in which only immediate collaterals are involved there is an excess of females, but not much greater than in all cases. They constitute 57 per cent. instead of 52 per cent.

We may now see, in part at least, why females preponderate so much more in the cases of inherited disease than when there is no inheritance. The inheritance is more frequently from the mother's side than from the father's side; when the inheritance is from the mother's side, females suffer in a much larger proportion than males.

Of the relatives who have suffered, the mother was affected less frequently than the father. The father suffered from epilepsy or insanity in 40 per cent. of the cases in which the inheritance was paternal; the mother herself in only 36 per cent. of the cases in which the inheritance was maternal.

Although the mother herself suffered less frequently than the father, the two suffered from epilepsy itself with equal frequency, and the difference depends chiefly upon the fact that the mother suffered from insanity much less frequently than the father. Of 762 cases of inherited epilepsy the father himself was epileptic in 87, and the mother in 91 cases; the father was insane in 36 cases, the mother in only 21.

In some cases the family tendency, as evidenced by the number affected, was very strong. In one case, for instance, five other individuals were epileptic—the patient's mother, maternal aunt, two uncles, and cousin. In another instance no less than fourteen members of the family suffered from epilepsy—the patient's mother and maternal grandmother; her mother's sister and mother's brother's son; four of her own sisters, and five of her sisters' children. In a third, eleven epileptic relations were known—the mother, both maternal grandparents, two maternal aunts, and of the twelve children whom the mother bore, the patient and five brothers were epilepic. In another instance the patient's father, two of the father's brothers, and three of his sisters were epileptic —another instance of six cases in one generation.

The neurotic diseases which can be traced in the antecedents of epileptics, and can be regarded as significant, have been already mentioned. Epilepsy and insanity are conspicuous, and epilepsy is the more frequent. A history of epilepsy was obtained in nearly three quarters of the inherited cases. In rather more than half (534) it existed alone; in 110 it was combined with insanity, in 8 cases with chorea, and in 3 with both insanity and chorea. Insanity occurred in the relatives of about one third (327) of the inherited cases, and of these it existed alone in two thirds.

not a certain, is often a probable indication of a morbid family tendency, and some weight must be given to it as an indication of a disposition to disease of which epilepsy may be a result, even when it has an immediate exciting cause. A lad whose first fit occurred during measles had two epileptic cousins on his mother's side, his mother's uncle and grandmother committed suicide, while three of the mother's cousins were insane. One patient's sister, insane, attempted suicide, and her mother's sister made a successful attempt.

The many allied diseases that are met with in relatives are probably sometimes related to epilepsy, but their general frequency and other relations reduce the significance of the association to a comparatively small degree. Chorea occurred in other relatives in 46 cases, in 32 of which it existed alone; in 8 it was associated with epilepsy, and in 4 with insanity. The forms of paralysis, paraplegia, and infantile palsy included in the investigation, were not more frequent in the families with other evidence of neurotic tendency than in those who presented no such history. The cases in which a strong family history of hysteria was obtained were very few. But there are many sources of fallacy. Hysteria is not common among the poor of this country, and is frequent among the better classes apart from other neuroses.

Heterogeneous Heredity; Indirect Inheritance.—Is epilepsy predisposed to by any other inherited tendency than that indicated by the occurrence of its own immediate allies? Gout, rheumatism, and phthisis have been supposed to favour its occurrence. Regarding *rheumatism*, apart from the heart disease which it may cause, it is very difficult to perceive evidence in support of the view. The influence of *gout* is equally difficult to discern. It is so common that most instances of concurrence are unlikely to be significant, and its relation can only be ascertained by a special investigation, on a very large scale, from both sides. The question will be presently considered. The effect of *phthisis* is also difficult to ascertain. It is certain that the family history of epileptics presents a large proportion of cases of phthisis, but the coincidence is probably accounted for by the commonness of lung disease. A relationship between the two is suggested by remarkable instances occasionally met with, but this is true of many other diseases. One patient, presenting no evidence of neurotic inheritance, had lost father, mother, and six brothers and sisters from phthisis, she herself, the sole survivor, being epileptic. The statistics which I can offer on this point are unfortunately not extensive; as far as they go they do not give support to the opinion that there is a relationship between the two diseases. Phthisis was inquired for in 300 cases, and a history of it in parents, grandparents, brothers or sisters, uncles or aunts, was obtained in 108, or 36 per cent. Considering the commonness of phthisis, this does not seem a larger proportion than might have been expected, although I am not aware that there are any statistics which can be taken as the standard of comparison. But if phthisis has any influence in causing epilepsy, we should expect to find it traceable more frequently in the cases without than in those with neurotic inheritance; but it is not so. Of 214 cases without, and 86 cases with neurotic inheritance, the proportion of phthisis is just the same—36 per cent. Further, if phthisis exerted any causal influence, we should expect to find the proportion in which a neurotic heredity is traceable smaller when there was a tendency to phthisis than when there was not. This also is not the case. The proportion was about the same in the cases with and those without a family history of phthisis. These facts suggest that the association of phthisis and epilepsy is accidental. The same conclusion is suggested by a search for a history of epilepsy in the families of the phthisical. My colleague Dr. F. Roberts has been so kind as to question on this point several hundred patients suffering from phthisis, and the proportion in which any relative was known to have suffered from fits was only one in forty. These facts seemed sufficient to make further investigation superfluous.

A similar conclusion is probably true regarding many other morbid states of which a history may be found in the parents of epileptics. Curious associations with every form of disease will be heard of, but this does not prove a causal relation to epilepsy, because instances as striking will be found in a corresponding number of individuals free from epilepsy. Nevertheless, sweeping statements regarding such causation of the disease have been made on the strength of instances of this character. In general, any causal relation to epilepsy of inherited diatheses outside the nervous system has yet to be proved.

There is, however, one constitutional state which comes into a conspicuous relation with some diseases not far distant from epilepsy, and has been thought to be distinctly related to epilepsy itself. It is the state which has been termed the "uric acid diathesis," and is closely connected with gout. The facts suggesting the relation have been insisted on, especially by Dr. Haig, but the facts are rather suggestive than conclusive. The frequency of the diathesis must be taken into consideration, and mere coincidence must be allowed weight. We must also remember that in an individual who is otherwise predisposed to epilepsy, and is already its subject, attacks may be excited by many influences which cannot be properly regarded as the causes of the disease. Inherited gout seems to be distinctly related to migraine, but such an analogy should do no more than make us observe with care, and on an extensive scale, recognising all sources of fallacy.

Inherited Syphilis.—In a few cases of apparently idiopathic epilepsy the patients were the subjects of well-marked inherited syphilis. All cases in which there were symptoms suggestive of local brain disease were left out of consideration. In spite of careful attention to the point for many years, I have been unable to perceive definite grounds for suspecting that epilepsy is a consequence of inherited syphilis, except as an indirect effect of its influence on general development. In very few of these cases did the attacks begin in infancy; in most they commenced towards the end of childhood, or after it. Any influence of syphilis on the nervous system, exerted indirectly through a toxic blood-state, would lead us to expect this result to be earlier and more frequent than it can be seen to be.

Maternal ill-health during pregnancy is met with in some cases. To it may, perhaps, be ascribed a share in causation, but it can only aid in an indirect manner. Whatever impairs nutrition hinders development, and the hindrance to the development of the nervous system may lead to convulsions in early life, which, in their turn, may dispose to epilepsy. Maternal "impressions" need only be mentioned as a curious illustration of the universal habit of noting the rare coincidence and ignoring the vast number of cases in which there is none.

Consanguinity of Parents.—The frequency with which relations marry is far less than is generally fancied, and is insufficient, in the case of epilepsy, to bring the consanguinity of parents into definite discernment. It may, however, have an influence when neurotic heredity exists in both parents. It is probable that consanguinity only intensifies existing tendencies, but sometimes mental defects, such as idiocy or blindness, may arise; very rarely epilepsy results. The following example shows that, apart from a morbid family taint, the marriage of relations may have a profound influence on the offspring. Two first cousins married, in whose families no nervous disease could be traced. Of seven children, four, three girls and a boy, were born blind from congenital defect of the optic nerve. Two other brothers had no defect of sight, but one of them, and also one of the blind girls, became the subjects of idiopathic epilepsy.

AGE.—The influence of age on the occurrence of epilepsy has been ascertained from the time of life at which the first fit occurred in a series of 3000 cases. The first fit, slight or severe, must be regarded as the indication of the time of the commencement of the disease. The frequency with which definite convulsive attacks are preceded, often for years, by attacks of "petit mal," renders much care needful, and has lessened the total number of cases from which trustworthy facts can be ascertained. Patients seldom suspect the relation of the slight attacks to those that are obtrusive, and often date the commencement many years after the actual onset.

If we take first decennial periods, we find that the number of (3002) cases in each period is as follows:

	Cases.	
Under 10	826, or	27·5 per cent. of the total number.
10—19	1398, „	46·5 „ „ „
20—29	463, „	15·5 „ „ „
30—39	186, „	6·3 „ „ „
40—49	71, „	2·4 „ „ „
50—59	40, „	1·3 „ „ „
60—69	15	·5 „ „ „
70—79	1	

The percentage of the whole constituted by the cases beginning in each period is shown in this table. Under ten years of age more than one quarter of the cases commenced, 27·5 per cent. Between ten and twenty nearly one half of the total number, 46·5 per cent., began. In the next decennial period, twenty to thirty, the number falls to about a seventh, 15·5 per cent. Between thirty and forty only 6 per cent. began, between forty and fifty about 2½ per cent., between fifty and sixty 1 per cent., and over sixty only ·5 per cent. of the total number. Thus 74 per cent. of the cases commenced under twenty years. This proportion (2224 of the 3002) is not far from that found by Hassex in the largest collection of cases previously made (757 out of 995).

The relation of the cases to age is shown in greater detail in the next table, which presents the annual number of 2383 cases commencing in each year up to twenty-one. After this age the numbers become too small and variable to make it worth while to give more than the smallest and largest number in each year of the decennium. The annual number is, however, given for the period after fifty-nine.

Age.	Cases.
Under 1	155
1	93
2	79
3	77
4	56
5	56
6	59
7	96
8	71
9	86
10	112
11	112

Age.	Cases.
12	149
13	149
14	183
15	166
16	167
17	133
18	144
19	93
20	79
21	65
22—29	9—30
30—39	3—17
40—49	1—13
50—59	0—7
60	2
61	1
62	5
63	2
64	2
65	1
68	1
69	1
71	1

A large number of cases, no less than 13½ per cent. of the whole, thus commenced during the first three years of life. In this group no cases of simple infantile convulsions are included, only such as, beginning in infancy, continued as chronic epilepsy. There is a difficulty in ascertaining the exact date of commencement in all cases. Of about one third all that could be learned was that they began in infancy. In the table these have been distributed through the first three years of life in the same proportion as presented by the two thirds in which the exact date of commencement could be ascertained. The number of cases is largest in the first year, and falls rapidly to three years of age, and then more slowly until five, when the minimum for the early period of life occurs. From this there is a considerable rise at seven, the commencement of the second dentition, then a fall at eight, and from this the numbers increase slowly at nine and ten, rapidly at twelve, until the maximum is reached at fifteen and sixteen, at which 166 and 167 cases commenced—about one seventh of the total number. From this period we have a rapid fall to twenty-one, after which only a small number began in each year.

The two series of cases, that given in the first edition, and chiefly drawn from hospital practice, and the second, largely from private practice, correspond so closely that it is not worth while to present them separately. For instance, in the first series the percentage between twenty and twenty-nine is 15·7, in the second 15·3. In the period between thirty and thirty-nine it was 6 in the first series and 6·5 in the second. A similar correspondence obtained throughout, and is evidence of the trustworthiness of statistical inference when the data are sufficiently large.

The influence of the epoch of puberty is shown by the fact that, during the years of age fourteen, fifteen, and sixteen, no less than 17 per cent. of the cases commenced. If the five years twelve to sixteen inclusive are regarded, they include the onset of more than one quarter of the cases.

Relation of Sex to Age.—The proportion in which the two sexes suffer varies considerably in the different periods of life. We may compare, first, their affection in the several decennial periods. (The general facts may be stated, without a numerical list.) In the cases commencing under ten years the females exceed the males by 8 per cent. In the second decennium, between ten and twenty, the excess of females amounts to 20 per cent. In the third, between twenty and thirty, there is a considerable fall, but the excess of females amounts to 12 per cent. Between thirty and forty, the numbers being still smaller, the relation is reversed; the males exceed the females by 19 per cent. Between forty and fifty, with a continued fall in numbers, the excess of males is still greater, amounting to 29 per cent. Between fifty and sixty it reaches 30 per cent., and over sixty males suffer almost exclusively.

If we examine the several periods of life more minutely, we find that in the cases which commence in the first year of life the females are almost twice as numerous as the males. In the second year the number of females falls considerably, while that of males rises slightly, so that the difference between them is less. During the two following years the numbers fall, until a minimum of each is reached at five years, the females being still in slight excess. At six and seven years a rise occurs, and the number of cases in each sex is, in the latter year, nearly the same. From seven to thirteen the earlier relation is reversed; the males are in excess of, or equal to, the females, with the perhaps accidental exception of the cases commencing at eleven. At twelve—that is in the thirteenth year of life—the maximum for males is reached, and from this until sixteen there is a slight fall. The maximum for females occurs later than for males. The numbers increase at twelve and thirteen, and then more slowly, until at sixteen the maximum for any year is attained, and they are in that year one third more numerous than the males. During the next five years the numbers in each sex fall rapidly, the females being still in excess, but the two are nearly equal at twenty-one. The females continue, however, in slight excess until twenty-nine, when the two sexes are affected equally. After this the relation between them is reversed—in almost every year the males are more numerous. The females present no increase at the climacteric period, and they are fewer in very late life than are the cases in males. Only one case commenced in a woman after sixty; in men, cases commenced at fifty-eight, fifty-nine, sixty-two, sixty-four, sixty-nine, and seventy-one.

Thus at each of the maximum periods, at infancy and puberty, the excess of females is very great. During the later period of childhood the numbers are nearly equal. The excess of females lessens after puberty, and ceases by middle life.

Relation of Heredity to Age.—How far does an inherited tendency influence the age at which the disease commences? It has been said that when there is heredity the disease begins before twenty. We shall, however, see that, so far from this being true, the influence of heredity continues until a late period of life, and, although absolutely greatest in youth, it is relatively only a little less during adult than during early life.

Particulars as to heredity and age at commencement were noted in 2222 cases, of which heredity existed in 888, or about 40 per cent. The distribution of these through life differed only slightly from that of the non-hereditary cases. About 5 per cent. began in the first year of life, and the annual number fell to a minimum in late childhood, to rise to a maximum at puberty. This maximum, however, occurred at fourteen years of age—a year earlier than the maximum for non-hereditary cases, which was at fifteen and sixteen. The cases with heredity lessen gradually during life, but continue to occur to the last. The latest case of the entire series was a man who suffered from what was apparently idiopathic epilepsy commencing at the age of seventy-one years, and in him the disease was inherited, his father having suffered from fits, clearly epileptic, for many years.

The relative frequency of heredity is shown by the proportion which the hereditary cases bear to the total number commencing at each period of life. The relation to age, in periods of twenty years, is shown in the following table:

Age.	Total cases.	Heredity.
Under 20 .	. 1592	661, or 41·5 per cent.
20 to 39 .	. 496	187, or 37·7 "
40 and over .	. 134	40, or 30 "

The average for the whole of life is about 40 per cent. This is exceeded by 1½ per cent. in the first twenty years; in the second twenty years the percentage is only 2·3 below the average; while in the cases commencing over forty it is only 10 per cent. below the average.

Relation of Heredity to Sex and Age.—Since the first twenty years include the two maxima at infancy and puberty, it is desirable to examine the influence of heredity during this period in greater detail, and we may at the same time consider how far the two sexes exhibit its effects.

During the first three years of life the disease is inherited by nearly the same proportion of females as of males (females 35·2 per cent., males 36 per cent.). During the next three years the proportion of heredity in females rises, but less than that in males (males 44·5, females 38). The third triennial period, six to eight, presents the same percentage of hereditary female cases (37·4), while that of males falls to 35 per cent. In the next triennial period, nine to eleven, the proportions are nearly equal (37 per cent. males, 38 per cent. females), and they are similar in the next triennial period, twelve to fourteen (36 per cent. males, 37 per cent. females). The difference becomes far greater between fifteen and seventeen inclusive, when the percentage of heredity is in males 30, in females 40 per cent. After this less difference is presented; in the next three years the percentage is 32 per cent. males and 38 females, and between twenty and thirty it becomes equal, 34 per cent. in each sex. Over forty, of 70 males there was heredity in 21 (30 per cent.); of 42 females there was heredity in 15 (nearly 35·5 per cent.).

These facts are surprising. In such a disease we should expect the effect of the depressing influences that act on the nervous system, as life goes on, in greater number, variety, and power, to throw heredity into the shade. The fact that it is not so is insignificant. We see that such causes do act in greater degree as life advances, but they are not effective, compared to predisposition, in the degree we should expect. It is also to be noted that heredity can be ascertained less and less as life advances and the preceding generation passes from the reach of questions. This brings out the fact that the disease is essentially related to congenital tendency, rather than to simple failure of nutrition and function, or to the many morbid influences of adult life. These may be effective, but the most powerful influence is the tendency to imperfect stability that is inherent in the nerve tissue, and is certainly largely dependent on inheritance. Its congenital nature renders it persistent long after the time when it might be expected to cease with completed structure and function. It will be considered more fully at a later page.

Post-epileptic Hysteroid Convulsion.—In a considerable number of cases of epilepsy an attack, which may be unnoticed, is followed by post-epileptic hysteroid convulsion, and its causal relation deserves note.

This immediate sequel is far more frequent in females, but the differences in the sexes is less the younger the sufferers. Under ten the sequel is met with in 15 per cent. of the male and 18 per cent. of the female cases. Between ten and twenty the difference is, as might be expected, much greater—14 per cent. of the male, and no less than 26 per cent. of the female cases. Between twenty and thirty the percentage of the males remains the same, and that of the females falls slightly. Between thirty and forty the male cases with hysteroid symptoms lessen considerably, while the females present a slight rise, and constitute 24 per cent. When epilepsy begins over forty this feature is absent in males; and in women it occurs chiefly when there is a suspicion of earlier attacks. In males the hysteroid sequelæ bear, at each age, nearly the same proportion to the cases of pure epilepsy, decreasing very slightly from a maximum in childhood to a minimum in middle life. In women, the minimum is in the first decade, the maximum is in the second; and the proportion after adolescence remains nearly double that of males during the period in which they occur. I think, however, that some surprise will be felt at the fact that, even up to the fourth decade of life, one third of the chronic convulsive cases presenting post-epileptic hysteroid phenomena occur in males. The surprise is lessened, however, when we note the peculiar features presented by the brain functions in these male cases, which often amount to an emotional instability resembling that of the female sex, probably connected with the original disposition to epilepsy. The surprise lessens also when we perceive the degree in which the epileptic process in the higher centres leaves the lower centres uncontrolled but energetic. The hysteroid convulsion resembles, at a different functional level, the post-epileptic automatic action to be afterwards described (also very common in males), and there is no sharp limitation between the state in which emotion explodes through random movement or escapes through ordered action.

EXCITING CAUSES.

We must regard, as the exciting cause of epilepsy, the condition or circumstance to which the first fit was apparently due. It may be again pointed out, to prevent misconception, that these exciting causes cannot be regarded as the essential causes of the disease except in a very small number of cases. They would have been ineffective had not a predisposition existed, due to inheritance, or to causes which are generally beyond our recognition. As already said, the exciting cause, as a rule, bears no more causal relation to the first attack than does a spark to the explosion of the gunpowder on which it falls. The existence of the predisposition is revealed by the action of the spark. Chemical attraction, restrained in unstable equilibrium, needs only the stimulus of the slightest added motion to cause sudden chemical change and the escape of the energy before restrained. The real cause of the disease is the morbid state of the nervous system, the existence of which is only revealed by the effect of the immediate excitant of the first fit. How potent is this element in causation, we perceive when we regard the large number of cases in which minor attacks occur first, often for years of so slight a character that no notice is taken of them. Nevertheless, since the occurrence of one fit undoubtedly facilitates the occurrence of others, we must regard the excitant of the first fit as, to some extent, the cause of the other fits, *i. e.* of the whole developed disease.

The study of these causes presents peculiar difficulty. The statements made by patients have, of necessity, to be received with caution, and the alleged facts to be carefully investigated. An occurrence so striking as a fit is naturally assumed to be due to some cause at least discoverable, and so is often attributed to a remote and improbable antecedent. Of 1665 cases in which this point was noted, a reasonable cause for the first fit was given in 696, or 42 per cent., and in the remainder no probable cause could be ascertained. From this series the cases distinctly originating in dentition convulsions are excluded.

Relation to Sex.—An exciting cause is met with in males rather more frequently than in females.

Relation to Age.—If we examine the proportion of cases in which an exciting cause can be traced in the different periods of life, we find that under ten it is nearly the same for both sexes. From ten to twenty there is a great difference between the sexes, excitants being traceable in many more males than females. In the next two decades, twenty to forty, the percentage in men continues nearly the same; but in women it rises in the first, and falls in the second. In the cases which occur over forty years of age the percentage in each sex is about equal (40); but in the male cases commencing over fifty an exciting cause could be traced in as high a proportion as two thirds. Thus the most striking peculiarity is the large proportion of cases which commence in women between ten and forty years of age without any discoverable exciting cause to which they can be ascribed.

We may now consider in more detail the various exciting causes that can be ascertained.

INFANTILE CAUSES.—Foremost among personal conditions is the fact that, in a very large number of cases, the attacks commence in infancy, during the first three years of age.

It is convenient to consider these cases together. They will be found to fall, for the most part, into three classes: (1) Those in which labour was protracted and difficult, or (seldom) premature and swift, in which the symptoms immediately after birth suggest some meningeal hæmorrhage. (2) Cases in which the attacks begin by a severe fit or series of fits, with indications of a local cerebral lesion. (3) Cases which constitute the majority, in which the infantile fits are at first slight, and are associated with retarded development, especially manifested by backward teething. There are, indeed, a few other cases in which some influence—exposure to the sun, or a fall, such as may be influential at any time—has preceded the first fit.

The largest group is the third, the cases in which epilepsy dates from the convulsions called "teething fits." In about two thirds of the cases beginning in infancy, particulars of which could be ascertained, the fits arose from such so-called "dentition convulsions." It seems legitimate to ascribe to the same cause a similar proportion of the cases respecting which no information was forthcoming. If so, we have a total number of cases due to this cause which constitutes 7 per cent. of all the cases investigated.

All now perceive the truth of the opinion, long ago urged by Sir William Jenner, that almost all convulsions associated with dentition are really due to the constitutional condition of retarded development which we call "rickets," from its influence on the bones, *i. e.* to the irritability of the nervous system which accompanies this condition. The further details of the cases fully confirm this, for in a large number there was a history of late teething and late walking, while many present crooked limbs. Doubtless an inherited neurotic tendency has some effect in disposing to convulsions under this exciting influence. Illustrations of this are occasionally met with. Thus infantile convulsions occurred in eight out of nine children whose mother's father and father's sister were epileptic.

Even these facts probably do not represent fully the influence of rickets in the production of epilepsy. In many of the cases just described the course of the attacks was continuous from infancy to adult life. In others, however, there were intermissions, especially during the period of childhood. In several, for instance, the fits ceased at seven, to recommence at ten or eleven years. In many other cases in which epilepsy commenced after the period of childhood was over, repeated convulsions had occurred during the first dentition, accompanied in most with other signs of rickets. It seems reasonable to ascribe to these convulsions of infancy a share in predisposing to the convulsions of later life, and the addition of these cases raises the proportion in which the infantile disease has some share in causation to as much as 10 per cent.

We speak of convulsions as due to rickets. But how are they produced? They coincide with the hindrance of development which occurs so often and so readily towards the end of the first year. Growth indeed goes on, but the functional development of the tissues is hindered; they do not present the capacity for function that should coincide. Any general illness or prolonged digestive disturbance may have this condition as a result. A severe attack of diarrhœa in a child of ten months may cause distinct signs of rickets.

How does the state of defective development cause the liability to convulsions? It can be adequately explained by the simple fact of retarded development. In the nervous system we know that, at birth, the state of the various structures differs,—that some are structurally perfect, while in others a considerable amount of developmental change has still to occur. Full structural development must precede full functional capacity. The nerve structures which are lower in function, and partly in position, are developed before the higher. This is true of the motor elements and reflex centres. The lower centres are under the control of the higher, and their activity in early infancy, when the higher centres are less developed, is manifested in the conspicuous activity of the reflex processes. Hindered development acts most on the least developed structures, those which should control the lower. Hence the tendency to spasm and convulsion in cases of rickets.

Whether rickets is or is not entirely preventable, the degree in which it leads to convulsions may always be prevented by proper attention to the diet and hygiene of infancy. These facts, therefore, suggest that a considerable proportion of the cases of epilepsy are really within the range of preventable diseases. But inherited taint exerts the same influence as at other periods of life, predisposing to the occurrence of convulsions in backward children, and to the persistence of the fits, and that the management of such children should be a subject of extreme care. Even a single fit in infancy indicates the need for careful supervision.

The second class is that in which epilepsy is the result of an organic lesion of the brain during infancy. These cases differ from those last considered in three features. The epileptic convulsions are one-sided, at any rate when moderate in degree; and if there is an aura, it is in some part of one side. The spasm can often be observed to commence locally in the hand or face. When both sides are convulsed, one is affected before the other, except in the most violent attacks. In these there may be no perceptible interval between the affection of the two sides. Inquiry regarding the character of the infantile convulsions will usually elicit the fact, when their character is known, that these also were confined to one side. The second feature is that the first attack of infantile convulsions was of great severity. Often, indeed, there was a series of convulsions, one after another, for several hours. Sometimes such a series was the only infantile attack; often such initial attacks were followed by others for a month or two, which then ceased, to recur when a few years had passed. Complete continuity of occurrence from infancy to adult life is less common in these than in the cases first considered. The third feature is that the first convulsions often occur during some acute illness, or soon after a fall, or in a state of general physical prostration. This is not always the case, nor is it always true that there are several consecutive convulsions at the onset. But the first fit is seldom slight in degree, and, if the facts can be accurately ascertained, the first fits will be found to have been unilateral.

In such cases there is often distinct hemiplegia at the onset, which may persist in some degree. They are considered in more detail at a subsequent page.

In a third group the first symptoms can be traced back, not only to infancy, but through infancy to the earliest period of separate existence. In some there were convulsions during the first two or three days of life; in others there were convulsive twitchings or some other indication, such as difficulty of swallowing, or inability to support the head, of damage to the brain. These cases occur in the children who are "firstborn," who enter the world with greater difficulty than those which follow them, whose birth is long and tedious, often needing the aid of instruments to terminate it soon enough to save the new life. Yet the same symptoms are met with occasionally in children who are born too early and swiftly, with cranial walls incapable of protecting the structures within from the slighter but sudden pressure to which they are exposed. In both classes the children often pass out of infancy with weakness of the legs, sometimes also of the arms, or with irregularity of the movements of the hands, and often also with convulsions. The symptoms are the result of damage to the cortex of the brain, commonly the effect of meningeal hæmorrhage, causing "birth-palsy." When the damage is slight there may be no symptoms of paralysis, but its effects are often manifested by the occurrence of convulsions, either in childhood or in later life.

In a few cases of epilepsy the fits can be traced back to infancy but do not fall into any one of these three classes. Most of these are associated with congenital mental defect, and occur in families with neurotic disposition, and in which other instances of idiocy can be heard of. In these the malady must be ascribed to a congenital imperfection of the nerve tissue, of which the convulsions and the mental defect are both consequences.

MENTAL EMOTION.—Of all the immediate causes of epilepsy the most potent are psychical—fright, excitement, anxiety. To these were ascribed more than one third of those in which a definite cause was given. Of the three forms of emotion, fright takes the first place. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life, when emotion is so readily excited, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of 173 cases only 14 commenced after thirty years of age, and 145 commenced under twenty. Of these the majority, 102, commenced between ten and twenty, only 43 cases before ten. The female sex is notoriously the more emotional, and accordingly the disease results from fright in a larger proportion of females than of males, although the difference is, perhaps, less than might be expected—61 per cent. of females and 39 per cent. of males. It is notorious also that this difference between the sexes increases as life advances. In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact. Under ten years of age the sexes suffer equally. Between ten and twenty the males suffer less than females as 3 to 4; between twenty and thirty as 3 to 13, and over thirty the only cases due to this cause occurred in women.

The influence of this cause on the motor centres is accurately recognised in popular phraseology. The stimulation of these centres causes the sudden muscular actions, such as the start of alarm, which originate in the need for immediate movement, which danger entails, through most of the animal kingdom. It is by instant movement that danger can be escaped or resisted. Fear is the great "e-motion," that which induces a "moving from," and, as a mental state, its effect has suggested names for a large class of mental sensations in such words as "tremble." If flight is impossible, the disturbance of the motor centres is shown by tremor. Such tremor may persist in the young as chorea, in the old as shaking palsy. In those predisposed to epilepsy it may leave a deranged state of function and nutrition which entails the sudden discharges of convulsion.

Predisposition usually exists. Hence, in speaking of fright as a cause of epilepsy, it must be remembered (as already stated) that its effect is only that of the exciting spark.

The exact form of fright varies, of course, in different cases, but the list is instructive. In several cases the cause was some stupid practical joke—as a pretended ghost, children shut up in dark cupboards, an alarm of fire, or burglars. In a few cases the patient had watched other persons in fits. Alarm during severe thunderstorms was another cause. One case was that of a soldier who had his first fit a few hours after being terrified, while on sentry duty at night, by the unexpected appearance of some white goats on the top of the adjacent wall of a cemetery, which he mistook for emissaries from the graves.

Of the cases in which the interval between the fright and the fit was noted, the fit occurred immediately in one third, within a few hours in one sixth.

Other forms of sudden emotional excitement may cause the first fit, and these cases also occur during the emotional period of life, under thirty years of age.

Prolonged mental anxiety is, in some cases, the only influence to which the disease can be assigned. In this series of cases it was given as the cause with apparent reason in 48 cases, in which males preponderated. This cause is effective late in life more frequently than most others.

TRAUMATIC CASES.—Next in numerical importance among the exciting causes of epilepsy are traumatic influences, blows and falls on the head. To these, after the elimination of doubtful instances, 108 cases were due. A third occurred under ten years, rather more than a third between ten and twenty. Males are affected by this cause more than females —63 to 45. This is clearly due to the risks of occupation, for in the cases commencing before ten the females were in excess. Between ten and forty the male cases in each decennium were rather more than twice the number of the females. Over forty the only cases were in men.

In four sevenths of the cases the injury was a fall on the head; in three sevenths it was a blow. In most of the cases the patient was stunned for a time, but in only a fifth did the first fit occur immediately. In the rest an interval elapsed; in one third of the cases the interval between the injury and the fit was more than a day and less than a week; in another fifth the interval was between a week and a month, and in about the same proportion the interval was more than a month.

It may be urged that these traumatic cases should not be included among cases not due to organic disease. But it is certain that a blow or fall may excite fits without causing any visible lesion of the brain, and all cases were excluded in which the present or past symptoms, or the mode of onset of the fits, made it probable that "coarse" changes had been produced.

Exposure to the sun is frequently assigned as the cause of the first fit, but the relation is often doubtful. Sunstroke affords so ready an explanation of a convulsive seizure occurring in the hot sun, that it is naturally regarded as the cause of the attack in all such cases. It is an undoubted cause in a few. After the elimination of all doubtful instances, in which some other cause could be traced, or in which the heat of the sun was not very intense, or the exposure prolonged, there remained thirty-five in which it seemed probable that the first fit was due to this cause. In several cases there was a distinct attack of sunstroke, occurring, in most, in the tropics. From this cause, as may be expected, males, from their greater exposure, suffer most. The cases were pretty equally distributed through life, but those in females were relatively most numerous in childhood, and decreased in adult life, to cease at thirty. In ten the fit occurred immediately; in several others, in which an interval elapsed, this was marked by some form of nerve disturbance, such as tremor.

Acute Diseases.—Recurring epileptic convulsions sometimes succeed acute specific diseases. In some of these cases the convulsions are distinctly "post-hemiplegic;" an attack of paralysis occurred, probably from thrombosis, during the illness, and this was succeeded by recurring convulsions. In other cases there is no history or indication of any preceding paralysis; the convulsions are apparently unconnected with any organic brain lesion. The diseases, in the course of or after which the first fit occurred, were as follows:

	Cases.
Scarlet fever	39
Measles	12
Fever—"low," "typhus," "typhoid"	8

A few cases were referred to rheumatic fever and diarrhœa, and to influenza.

The remarkable influence of scarlet fever in the causation of epilepsy demands further consideration. The cases due to this cause commenced during early life, under eighteen. It may be thought that the first fits in these cases were due to uræmic poisoning, but this is not supported by the particulars ascertained. In most instances there was no evidence of renal disease; the fits commenced either during the acute disease or during uncomplicated convalescence. In many instances the initial convulsions were succeeded by attacks of petit mal, which changed to severe fits only after some years. In a few cases convulsions ushered in the disease, persisted throughout the illness, and continued afterwards as permanent epilepsy. In only one case did ear disease also result from the fever. The urine was examined in most cases, but no albumen was found.

These facts suggest that the first fits w
peculiar effect of the scarlet fever

system. It may be remembered that this specific poison has a very wide-spread action, and that after scarlet fever, optic neuritis, leading to subsequent atrophy, may occur without renal or cerebral disease.

The cases are rare in which epilepsy is the result of other acute specific diseases, with the exception of influenza. We have learned, however, that the organisms which cause the symptoms of these diseases give rise to chemical changes in the body, and thus produce, directly or indirectly, toxic material which has often a special affinity for the structures of the nervous system, and deranges their functions. Such an influence and its consequences must be carefully distinguished from the occasional effect of the disease in causing a local cerebral lesion, already referred to, which may cause convulsions either as its initial or ultimate effect.*

REFLEX CAUSES.—Irritation of peripheral nerves, visceral or external, is occasionally the exciting cause of convulsions, which may continue as persistent epilepsy. This is much less frequently the consequence of irritation of the cerebro-spinal nerves than that of the gastro-intestinal nerves, in spite of the fact that the latter seldom cause sensations which reach consciousness, while the former usually induce obtrusive pain.

Peripheral irritation in the cerebro-spinal system can be traced as the apparent cause of the first fits in an extremely small number of cases. Especially rare are those in which the symptoms of the attack afford confirmation of their apparent cause. Indeed, cases which are apparently thus produced often turn out to be cases of central disease in which the peripheral cause has been only a minor excitant, so as to justify considerable scepticism regarding the influence of this agency on the normal brain. For instance, a man had his left forearm painfully bruised in an accident. A few days afterwards he had a convulsive attack, which began in the injured limb by pain similar to that felt after the accident. Such attacks were repeated, but headache supervened, and acute optic neuritis was found to be present. The patient had had syphilis, and treatment for this quickly removed all symptoms. The injury to the arm had merely served to excite attacks, the real cause of which was the organic brain disease, a syphiloma adjacent to the opposite motor area. In other rare cases in which peripheral irritation seems to excite convulsions we may reasonably assume a morbid instability of the centres, although not dependent on organic disease. A girl ran a spike into the palm of the hand. Prolonged pain resulted, and six months later she began to have attacks, which commenced by a feeling of tingling at the seat of the injury. The tingling spread down the fingers and up the arm to the face, when there was loss of consciousness and clonic convulsion. The local pain, however, had ceased when the attacks set in. Again, a lad of fifteen, working as a smith, got a fragment of steel embedded in the right cornea, which caused great pain. A comrade tried to extract it with a pointed piece of wood. As soon as this touched the eye he felt a thrill through the system, the right orbicularis began to twitch, the head turned to the right, and the right limbs became convulsed. Other fits followed during nine years, affecting, according to degree, the upper part of the face, or this with the head, or the whole right side, with loss of consciousness.

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* The influence of local cerebral lesions during acute specific diseases has received undue weight from some French writers (*e.g.* Marie, 'Prog. méd.,' 1887; Lemoine, ibid., 1888), who infer that general convulsions may be a result of cicatricial changes about a "bacterial" lesion. But a focal change has local manifestations, and without these we are not justified in its assumption.

In other cases it seems as though the pain has a wider influence, exciting general convulsion without local symptoms related to the source of the pain. In such cases there is usually evidence of the existence of a definite predisposition. A girl, with a strong family history of epilepsy, had her first fit five hours after the extraction of a tooth which had caused severe pain. In another patient, who had suffered from fits in infancy, severe toothache coincided with the first epileptic fit at thirteen. In a case in which there was not only inheritance, but also a history of infantile convulsions, the first fit occurred at sixteen during intense pain produced by suppuration in the middle ear, without evidence of intra-cranial complications. More directly significant of peripheral excitation is the case of a patient, also with a family history of epilepsy, who, after a severe blow on the nasal bone, became subject to distinct minor attacks, consisting of an unpleasant smell, with giddiness, lasting a few moments. The attacks always occurred in the early morning, in a characteristically epileptic manner. Less weight may perhaps be given to a girl of fourteen, also with heredity, and having had much mental anxiety, in whom the first attack was induced by a friend persistently tickling her feet—a procedure which must, however, have a profound influence on the nervous system if the statement is true that it is an occasional mode of executing women in China.*

Gastro-intestinal irritation is an occasional cause of convulsions, often without any pain to indicate the irritation of the nerves of the viscera, which must, nevertheless, be the immediate mechanism. Attacks may result from the presence of the larger forms of intestinal worms in children, and sometimes in adults. Usually, however, they cease when the worms are expelled. In rare cases the attacks, set up in the first instance by the intestinal irritation, recur and continue after the irritation is at an end. In eleven cases of this series the first fit was apparently due to this cause; the attacks had continued, although the worms had been expelled, and a renewal of the vermifuge treatment had no influence.

Digestive derangement is an occasional cause, but is only an excitant, effective on account of the predisposition of the nerve centres. When the first attack follows a very anomalous and indigestible meal, this may reasonably be regarded as the immediate cause, but a recurrence takes place sooner or later in such cases, as a rule, without any similar excitant. The influence of this cause is occasionally manifested by cases of organic disease, which augment the central excitability. In a child an attack of convulsions was distinctly excited by swallowing a piece of slate pencil, but a few weeks later indications of organic disease appeared and increased, the result, as was found post mortem, of a tumour of the pons. In many cases of tubercular meningitis the first symptom is a convulsion apparently induced by an indigestible meal.

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* To these cases Nothnagel proposes to give the name "secondary epilepsy" instead of "reflex epilepsy." They usually result from injuries to nerves. In some cases the effects appear purely functional, but it is possible that in some cases neuritis plays a part in producing the result. Among recorded cases are—Virchow: shot wound of median, followed by delirium, epileptic attacks, and mental derangement. An inch of the median nerve was excised, in which Virchow found proliferating neuritis, and the attacks and derangement passed away. But cf. the following:—Billroth: injury to sciatic; epilepsy; the nerve was cut down on, appeared normal, and was left alone, and the attacks ceased. Dieffenbach: injury to hand by splinter of glass; epilepsy; excision of scar; epilepsy ceased; nerve at spot thickened and hardened. Eccheverria: nervous girl of fifteen fell on right elbow; epileptic attacks, and symptoms pointing to a lesion of the ulnar nerve; after four years two inches of the nerve were excised, and found to present distinct neuritis; no more fits when published—only, however, four weeks later.

MISCELLANEOUS CAUSES.—*Asphyxia.*—In a few cases immersion in the water caused the first fit. This is an influence which involves both fright and asphyxia, but the influence of the latter was well shown in a child æt. 3¼ who tried to swallow a large piece of potato, which stuck in his throat and stopped his breath. He became livid, unconscious, and convulsed before the obstruction was dislodged with a spoon. The convulsive twitching continued for some minutes, and twenty minutes passed before he regained consciousness. Three days afterwards he had another fit, and they continued until he came under treatment, several months later.

Chronic alcoholism is an occasional cause of epilepsy.* Convulsions not unfrequently result from it, but usually in association with distinct symptoms of chronic meningitis. Now and then, however, simple epileptiform convulsions may result, and continue, without other sign of brain mischief. It was the apparent cause of 29 cases. In some the first fit occurred during intoxication, and in most the attacks recurred after each alcoholic excess, and only then. Epilepsy from absinthe-drinking, occasionally met with in France, is unknown in this country.

Lead Poisoning.—Epilepsy seems occasionally to result from chronic lead poisoning. In some cases there is also chronic renal disease. In one, epilepsy had existed from infancy, but became much more severe when the plumbism was developed. In the remainder, the lead poisoning was the apparent cause of the attacks, which resembled in character and course those of idiopathic epilepsy. It is well known that acute lead poisoning may be accompanied by convulsions; and that chronic poisoning may cause chronic convulsions precisely like those of ordinary epilepsy has also been pointed out by others. The list of nervous disturbances which may result from lead is a long one, and includes, as cases I have seen testify, acute and chronic mental derangement, neuralgia, brachial spasm, as well as palsy, general muscular wasting, and optic neuritis. It is known that lead is to be found in the brain in considerable quantity after death.

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* The conclusions from my own experience do not accord with those of Eccheverria ("On Alcoholic Epilepsy," 'Journal of Mental Science,' Jan., 1881).

Renal Disease.—I have mentioned that in two of the cases of lead poisoning there was also consecutive renal disease. In two other cases of chronic renal disease convulsive attacks, resembling perfectly idiopathic epilepsy, brought the patients under treatment. In one the absence of any coarse changes in the brain was proved post mortem. It appears, therefore, that such convulsive attacks may occur and recur for months, without other sign of uræmic mischief. Several cases in which chronic mental derangement resulted from Bright's disease have also come under my notice. Many other facts suggest that the disordered metabolism involved in renal disease may result in the production of various abnormal substances, some of which may have a special influence on nervous structures.

Tobacco.—I have met with no case which suggests that tobacco smoking exerts any influence in the production of epilepsy; but in one case the fits were apparently due to the patient, a lad, having shortly before commenced working in a tobacco factory, where his occupation caused frequent nausea.

Anæsthetics.—In two cases the first fit occurred after the administration of chloroform, and, in one, attacks, which had ceased for many years, recurred after the inhalation of nitrous oxide. It is remarkable that when the anæsthesia from nitrous oxide is passing off, transient hysteroid convulsion frequently occurs. The legs and arms are stretched and stiff, sometimes quiver; the patient looks wild, talks for a moment as if dreaming, then passes into sleep for a few minutes and is better.

Disturbed Menstruation.—Retarded or absent menstruation coincided with the first fits in a large number of the cases which commenced in girls between fourteen and seventeen, but the difficulty in determining the exact causal relationship between the two conditions is very great. When epilepsy is once set up in such cases, the subsequent establishment of regular menstruation appears to exert very little influence upon the fits beyond the fact that they are rather more likely to occur at the menstrual period, however regular this may be in time or normal in its character.

The period of puberty in girls is a time at which epilepsy is known often to commence, or to become more severe. Facts give no countenance to the common hope that preceding epilepsy will cease at the establishment of the catamenia, but rather indicate the reverse—that the attacks are least likely then to stop. The period is one of general disturbance of the nervous system which seems to favour the occurrence or persistence of the disease.

Pregnancy.—The first fit occurred, in ten cases, during pregnancy, without other obvious cause, and in five cases the disease commenced after parturition. In one case of this onset the attacks occurred during each of five successive pregnancies, only during sleep. They then became constant, and occurred also during the day. (See also "Course.")

Masturbation.—Many circumstances render it very difficult to determine the influence of masturbation as a cause of epilepsy. The habit is common in epileptic boys, as in others, but we cannot infer that, in all such cases, it is the cause of the disease. The etiological relation can only be regarded as established when the arrest of the habit, as by circumcision, arrests the disease. But the converse is not true; the continuance of the disease after the arrest of the practice does not disprove the relationship, because, when the "convulsive habit" is established, it frequently persists after its cause has ceased to be effective. Moreover, in private it is often difficult to ascertain the existence [illegible] ctice, for it is remarkable how long it may elude detection. It is usually denied by the patient, and the very inquiry renders its discovery more difficult by suggesting the necessity for concealment. I am inclined to think that it is much less frequently a cause of true epilepsy than of vague nervous symptoms and of untypical attacks, sometimes hysteroid, sometimes of characters intermediate between the hysteroid and epileptoid form. It is a frequent cause of severe "habit-spasm" developing in boyhood.

Syphilis.—Convulsions are very common in cases of syphilitic brain disease, syphilitic tumour, and chronic meningitis. Such convulsions may recur in chronic course, and may persist after the original disease has been rendered quiescent by treatment. The cases which present them are often called "syphilitic epilepsy." But they differ pathologically from the cases to which the term "epilepsy" is strictly applicable, in that post mortem there is found to be visible organic brain disease—chronic meningitis or a cicatricial growth. In most cases the convulsive attacks have the deliberate march and limited range characteristic of those due to organic brain disease, of which there are usually other indications.

Does syphilis cause epilepsy independently of the agency of organic brain disease—by such an action of the syphilitic poison upon the nervous sysem as eludes discovery by the most careful naked-eye and microscopical investigation? Fournier* has maintained that it does; that in the early period of constitutional syphilis a morbid state of the nervous system is induced by the influence of the syphilitic poison, and that this morbid state may be manifested by various functional derangements, as epilepsy, hysteria, and chorea. He asserts that "secondary" epilepsy is without visible lesions, while "tertiary" epilepsy results from organic disease. Stated thus, the assertion is unquestionably inaccurate. My own experience entirely agrees with the statements of Echeverria,† that organic disease, especially meningitis, is not rare in the early period of the constitutional disease, and that in most cases of convulsions occurring during the "secondary" period symptoms of such organic disease may be found. A few cases commencing in the early stage have the aspect of idiopathic epilepsy, but in most of these, other causes, such as inherited predisposition, can be traced; and it seems unjustifiable, in an inquiry into causes, to regard the preceding syphilis as more than a coincidence in such cases.*

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* 'Ann. de Dermatologie et Syph.,' 1880, pp. 160, 199.

† 'Journal of Mental Science,' July, 1880.

There remain, however, a very few cases in which no other cause can be traced, and in which, when iodide of potassium alone is given, the convulsions cease. Most of the cases which have been recorded in proof of the existence of such true syphilitic epilepsy are inconclusive: either the symptoms of the case, and the mode of onset of the fits, are so imperfectly described that the existence of organic disease cannot be excluded; or there existed other sufficient cause for the attacks; or bromide of potassium was combined with the iodide, thus depriving the effect of treatment of its significance. Cases which are not open to these objections are extremely rare, and I have only met with one. It was that of a man aged 37, who had a primary sore a year previously, subsequently sore throat, and presented traces of past iritis. There was no neurotic family history. Nine days before being seen he had a severe fit, without warning, in which he bit his tongue. There was no headache, ophthalmoscopic change, or other indication of organic brain disease. Ten grains of iodide of potassium, three times a day, were ordered, and four months later, when last seen, he had had no recurrence. Even such a case, however, is rendered scarcely conclusive by the fact that occasionally organic brain disease may cause no symptom but convulsion; even headache may be absent; and such convulsion may have the sudden general character met with in idiopathic epilepsy. The question must thus be regarded as still an open one.

The associations of epilepsy with heart disease, with chorea, and with past inactive organic disease of the brain, are considered in another chapter.

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* It is not denied that if constitutional syphilis acts as a cause of epilepsy independent of organic disease, it may be effective in patients with inherited tendency, the disease being the result of both influences; but in an inquiry to ascertain the facts such cases should be excluded. I have met with no other cases which suggest that syphilis itself causes idiopathi

CHAPTER II.

SYMPTOMS.

GENERAL CHARACTERS OF EPILEPTIC FITS.

THE attacks or seizures which characterise epilepsy are commonly divided into two classes,—major or severe, and minor or slight. These two forms, although clearly distinguished in their general characters, are not separated by a sharp demarcation. In the major attacks (*grand mal*) there is loss of consciousness, often prolonged, and severe muscular spasm. In the minor attacks (*petit mal*) there is commonly brief loss of consciousness without muscular spasm; sometimes there is loss of consciousness and slight muscular spasm; very rarely there is slight muscular spasm without loss of consciousness. The French term "petit mal" has become widely used, but the slight attacks it designates are termed by their subjects "sensations," "turns," "faints," &c.*

In severe attacks the patient, if standing, falls to the ground; in slight attacks he may or may not fall. But the fall has always been recognised as the characteristic feature. The term "epilepsy" embodies the mythical pathology that is also preserved in our word a "seizure," and is less simply descriptive than the old English name, "falling sickness," met with in print as far back as 1200.

In very severe attacks muscular spasm comes on at the same time as the loss of consciousness. In less severe fits (especially from local disease) muscular spasm may commence before consciousness is lost, and the patient is then aware of the onset of the convulsions. Still more frequently the spasm and loss of consciousness are preceded by some sensation. It may be referred to one of the special senses, or to some part of the limbs or the head, or may be an actual movement. The sensation or the commencing spasm, which informs the patient of the oncoming attack, constitutes the "warning" or "aura" of the fit. Its nature and features will be subsequently considered.

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* When an epileptic patient is first seen the physician should ascertain by what word the attacks are designated. A habit should be carefully cultivated of never using the word "epilepsy" or "fit" in the presence of a patient without ascertaining that it is familiar. "Attack" or "faint" is always available.

MAJOR ATTACKS.—At the onset of the severe fit the spasm is tonic. There is rigid, violent muscular contraction, which develops in the course of a few seconds, and fixes the limbs in irregular postures. The head and eyes are usually turned towards one side. The rotation may involve the whole body, and may even cause the patient to turn round sometimes more than once. This tonic spasm involves all the muscles, even those of the chest and abdomen. The features are distorted; the face, at first unchanged, becomes flushed and then livid, as the chest is fixed and respiratory movements are arrested. The eyes are open or closed; the conjunctiva is insensitive; the pupils, at first unchanged, dilate as cyanosis comes on. The limbs are fixed in postures which are generally those of imperfect extension, but not quite the same on the two sides. The variations will be presently described. As the spasm continues, it commonly alters in its relative intensity in different parts, so that changes in the position of the strained limbs occur. Presently, when the cyanosis has become most intense, in the fixed tetanic contraction of the muscle, slight vibrations can be felt, and these increase gradually to visible remissions. As these remissions become deeper the muscular contractions become more shock-like in character, and the stage of clonic spasm is reached, in which the limbs, head, face, jaw, trunk, are jerked with violence. Energetic movements of the chest occur, air is expelled from the thorax, and bloody saliva is frothed out between the lips. The air entering the lungs is at first insufficient to lessen the lividity, and the patient may seem to be at the point of death. But as the remissions become greater, and longer in duration, more breath enters the chest, and the lividity lessens. In becoming less frequent the muscular contractions do not become less strong, and the last jerk is often as violent as any of those which have preceded it. At last no other jerk occurs, the spasm is at an end, and the patient lies senseless and prostrate, usually sleeping heavily for a time. Urine frequently, and fæces occasionally, are passed in the fit.

This is, however, only an outline of the commonest type of severe fits. In attacks of less severity, yet purely epileptic, the variety in the features, in the character, seat, and spread of the motor disturbance and its preceding sensory disturbance, is extremely great. Some conception of these variations will be gained from a perusal of the features of the initial symptoms of attacks and the sensory warnings. To understand the variations it is important to remember that the convulsion is the result of the sudden release of nerve impulses in the motor cortex of the two hemispheres. These share, in approximate degree, the alteration of nutrition which promotes such discharge. The change may, however, be greater in one side, or in one part of one side. In some cases of idiopathic epilepsy, and in the large class in which a local lesion has set up fits, the excitability is excessive at one spot. But the connection between all the cortical structures in each hemisphere, and also between the corresponding structures of the two hemispheres, is so intimate that discharge spreads according to its degree of intensity, as well as according to the degree to which the structures to which it spreads are predisposed to respond to it. In the cases in which there is no general disposition, forms of slow discharge are met with, due to local instability, and resembling those due to organic disease, to be afterwards described.

The spasm and its extension are thus deliberate in onset. Instead of commencing simultaneously in all the muscles of the body it begins in one region, as the face or arm, and then spreads, first to the limbs on the same side, the head and eyes being turned towards that side, and then, lessening on the side first affected, it invades the limbs on the other side, with a corresponding rotation of the head. Such attacks may commence with tonic spasm; but frequently they commence with, and consist of, clonic spasm only. In such cases consciousness is often lost late, so that the patient is aware of the commencing spasm.

MINOR ATTACKS.—The slight attacks may consist of loss of consciousness only. An individual suddenly stops in his occupation, looks strange for a moment, perhaps turns pale, and then goes on with what he is doing, may even finish a sentence which he had commenced, and be aware that something has happened only by finding that he has dropped what was in his hands, or that persons near are looking at him in surprise. Such an attack is, however, often heralded by some sensory warning or aura, such as precedes the major attacks. It may be accompanied by slight visible spasm, such as putting the arms forward and bending the head down, or there may be slight convulsion in the part in which the spasm commences in the severe attacks, and, in some cases, the minor attack may be constituted by such spasm, without loss of consciousness. After a slight attack is over the patient may be quite well. Often he is stupid and dull for a time, and sometimes proceeds to perform some action in a dream-like, automatic manner, such as undressing himself, retaining afterwards no recollection of what he has done. Sometimes this stage is marked by passion and violence, even of the dangerous intensity designated "epileptic mania."

After an attack, major, or more frequently minor, instead of presenting an automatic state, the patient may pass into a condition of hysteroid convulsion, such as has been already mentioned, and will be more fully described. In some patients in whom this sequence occurs, all the obtrusive phenomena of the attacks may be of this hysteroid character, and the case is regarded as one of hysteria, or as a diagnostic refuge, capaciously vague, "hystero-epilepsy." They are cases of true epilepsy in persons disposed to hysteria, in whom the epileptic fit is followed by hysteroid disturbance.

SYMPTOMS IN DETAIL.

The symptoms of attacks may now be considered in greater detail. In describing them, however, it will be well to point out some of the physiological and pathological facts which underlie them, where these can be traced. By doing so the symptoms themselves are rendered more intelligible, and their study more instructive, than if they are merely enumerated without reference to that which we can conjecture regarding their pathology and pathological physiology. The explanations here given involve some physiological assumptions which find their justification in the explanation they afford of that which would otherwise be unintelligible.

In discussing the symptoms of idiopathic epileptic fits, it is necessary to allude to the characters of the analogous convulsive seizures which result from such organic diseases of the brain as tumour. These facts are, indeed, of supreme importance. It will, however, be understood that none of these cases of active organic disease are included in the statistics given.

Precursory Symptoms.—Beside the immediate warning, attacks in some patients are preceded for hours or days by symptoms indicative of disturbance of the nerve centres, and by these the patients and their friends know that an attack is impending. These "prodromata" (as they may be termed in distinction from the immediate warnings) are of two classes,—sudden momentary symptoms, recurring frequently, or some continuous indication of slight disorder.

Paroxysmal Prodromata.—The brief momentary recurring symptoms that occur for a few hours or a few days before a fit seem to be essentially of the nature of minor attacks. Patients who are liable to minor seizures may have them chiefly in this manner for a day or two before the severer attacks. As a rule, the recurrent symptoms referred to are slighter than such as are usually regarded as petit mal, although, as we shall see, true minor attacks may be as slight.

Among these prodromata, one that is not uncommon is a brief "dreamy state," or a sense of strangeness, occasionally with definite unconsciousness, recurring many times.

Another, also frequent, is the occurrence of sudden starts or jerks, affecting both sides, sometimes the arms only, sometimes the whole body and limbs. The jerk is occasionally in one arm only, although the fit is general and the malady idiopathic. The patient may, in the jerk, drop anything that is in his hand, or even, when the legs are involved, may fall down. These jerks occur in some patients for an hour or two, in others for three or four days or even a week before a fit. They are usually limited to the waking state, but in rare cases occur only during sleep, or during the state of "going to sleep."

Among other recurrent symptoms of this general character are sudden momentary epigastric pain, a sudden brief sense of dyspnœa or of choking, hiccough, palpitation of the heart, and brief recurring local pain in the head. In one patient, whose epilepsy dated from a fright at three years old, each attack was preceded for about an hour by a sensation of "pins and needles" and "pricking," first across the back of the right wrist, and then passing down to the fingers, and afterwards felt at the back of the metacarpal bone of the left thumb. It was somewhat painful, and continued until the fit came on.

Giddiness, occurring in brief paroxysms, seldom intense, may precede attacks. The vertigo is often distinctly of the nature of petit mal. Sometimes it is more continuous, as in a patient in whom a sense of a movement of objects to the right existed during a day before an attack, but just before the onset there was intense subjective vertigo, a sense of spinning round to the left, with actual movement in the same direction, and also nausea with vomiting. Various other forms of paroxysmal disturbance are also met with. One patient always had severe attacks of sneezing for a few hours before a fit. Yawning is another occasional prodroma.

In one instance of yawning attended by nausea, there was always vomiting after the attack.

Continuous Prodromata.—A peculiar irritability, sometimes with lethargy, occasionally exists for a day or two, and may continue until the fit, or may be replaced for an hour or two by brightness and cheerfulness. A more profound mental change may herald an attack, even for a week, and may increase in amount until the attack—excitability and even delusions. Now and then there is some change in the aspect of the patient, especially a vacant look, which is recognised by the friends. Somnolence for a day or two is sometimes a premonition.

Occasionally the precursory symptom is an unnatural appetite for a day or two; the patient eats ravenously, and never seems satisfied, and from this it is known that an attack is impending.

Headache is a common precursory symptom, seldom local (as at one temple), usually general and dull. It may be accompanied by mental dulness, but commonly exists alone, for an hour or two, or even longer, even for three or four days. Some other cephalic sensation may precede the attack. Occasionally (as Aretæus remarked) flashes of light or colour are the precursory symptoms, as in a case in which the fits began in the leg and were preceded for some hours, or even days, by occasional "sparks of red fire" before the eyes. In such cases we have a clear type of minor attacks. The sudden startings above mentioned may be accompanied by bright lights, or the latter may occur alone. There may be frequent momentary attacks of loss of sight, and each, in one case, was followed by a bright flash during the hour before the fit, the immediate aura being simply darkness. An auditory prodroma was met with in one case only—a man of sixty-two, epileptic since forty-seven, with bilateral labyrinthine deafness. There was no immediate aura, but a sound of bells ringing was heard for a day previously, and persisted for a time after he woke from the subsequent sleep.

In rare instances the onset of the fit is preceded by some automatic action, as running. A patient may always run a short distance, and then fall in the fit. It is probably sometimes associated with the emotion of fear, of which rapid movement is the natural effect. The emotion may not be remembered, but it is sometimes suggested by a scream, which is not the mere laryngeal epileptic cry, but is distinctly that of fear. In one patient, a boy, the cry accompanied an epigastric aura and a sense of falling; he ran across the room, then screamed as he fell in the tonic spasm of the fit. These cases were termed "epilepsia cursiva" by Boötius.*

Another patient, whose attacks were epileptic and severe, if walking, always turned and walked back a short distance, and then fell in the fit. He turned back in the same manner although he was going home, and near home. This is probably a more elaborate result of a mental process, which could not afterwards be recalled.

The face usually becomes pale after the fit comes on, but not before it. It can seldom be observed in severe attacks, because the congestion from fixation of the chest comes on so quickly, and the aspect is afterwards determined by the mechanical effect of the spasm. In minor attacks the aspect can be better observed; it may be unchanged, or pallor may coincide with the passing away of the attack. When the face is pale at the beginning of the fit, this is often accompanied by an expression of fear, although no warning is remembered, and may indicate an initial mental state, the effect of which is not such as to permit its reproduction in "recollection."

This automatic action at the onset of fit is important as showing that the process is not, as has been said, purely post-epileptic. It may be a manifestation of the commencing cerebral discharge occurring in a special manner, and culminating in the ordinary form. It is comparable, on a higher level, to the local commencement of a fit by spasm in one arm followed by loss of consciousness. It deserves note, moreover, because, in rare cases, certainly epileptic, to be presently described, all the convulsive movement is of a similar "co-ordinated" character. In another example of such pre-epileptic automatic movement, after a sense of falling, with an epigastric sensation and a scream, the patient rushed across the room, then gave another scream, and fell in the fit. The "rushing" in this case may be regarded as the mere result of an initial emotion; but this, and many other warnings, are the result of an elaborate process which has its motor counterpart in automatic movement.

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* In 'Observationes Medicæ de Affectis Omissis,' London, 1649. Several instances were described by Andrée in 'Cases of the Epilepsy,' &c., 1746.

MODE OF ONSET—WARNING—AURA.

The first indication of an attack yields information of great importance, practical and pathological. The convulsions of idiopathic epilepsy are so rapid in their evolution, and so similar, in most cases, in their developed characters, that mere observation of attacks was for a long time unproductive of much addition to our knowledge. A new era in the study of epilepsy may be said to have commenced with the investigation, by Hughlings Jackson,* of the mode of onset of attacks which begin deliberately, a study of the place in which the convulsive spasm begins and how it spreads. This has been supplemented by an extensive series of facts regarding the sensations with which attacks begin. A sensory onset is far more common in cases in which the attack is sufficiently deliberate for the first symptoms to be observed and to be retained by consciousness.

In considering this subject it is important to distinguish, but also to compare the cases in which the attacks are due to an organic lesion of the brain, or are "idiopathic." Many pathological facts have been ascertained regarding the former, and the generally uncertain indications of these have received important extension, and more precision, by the results of experiment, confirmed by the facts ascertained in the course of operations on the brain.

The aura of an attack is the sensation, usually in some part of the periphery, with which attacks commence. Even when it is muscular spasm the subject is conscious of it by the sensations produced. A sensory "aura" was formerly renected, directly or indirectly, with a part concerned with motion, may be regarded as a proposition scarcely needing proof. A discharge, such as occurs in an epileptic fit, taking place in either of these related structures, may remain confined to that in which it commenced, but it usually leads to a discharge in the other. The two may act so simultaneously that the resulting motion and sensation occur together. The patient may feel a tingling in his hand at the same moment as the fingers begin to twitch. Or the discharge in one may lead the way—to be followed by the other at an interval. The twitching or tingling may come first. It is of importance to determine, in any given case, with which the attack commences—the motion or the sensation,—*i. e.* whether the motor or the sensory centre leads in the discharge. By so doing we are able to understand better the association of auras and their progress. It is necessary, however, carefully to discriminate the form of the sensation, because, as Hughlings Jackson has also pointed out, the consciousness may be affected directly by the action of a motor centre, although no movement results. There are motor sensations as well as sensory sensations, and, as we shall see, a fit may begin by a sense of motion before any actual movement occurs, and the sensation of motion may be vastly in excess of the real movement. The word "sensation," however, will here be used, when unqualified, to signify a sensation proper, such as numbness, tingling, and the like.

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* Especially in "A Study of Convulsions," 'Transactions of the St. Andrews Med. Graduates' Association,' vol. iii, 1870; and "Localisation of Movements in the Brain," 'Lancet,' 1873, vol. i; also republished separately.

Association of Warnings.—One sensation may be followed by another, and this by a third or fourth, and some may be simultaneous. These associations are especially frequent and instructive in the forms in which the special senses are involved. In them, especially when uniform or frequent, can often be traced indications of physiological relations, and such are occasionally suggestive of even more than we can yet clearly discern.

Frequency of Warnings.—In what proportion of cases is consciousness lost so early that the commencement of the fit is unfelt? Statistics must be based on the record of presence or absence, and it is certain that absence will escape record far more frequently than presence. The positive is conspicuous, the negative is not, and needs an effort for such recognition as ensures record. In the first series the notes recorded presence or absence in nearly two thirds, and an aura was present as frequently as absent. In the second series it was noted in nearly the same proportion, but was present twice as frequently as it was absent. This may have been due to the fact that the patients had a much greater average degree of intelligence. It should be noted, moreover, that some patients mention a warning at the moment of the onset of a fit, but are quite unable afterwards to recollect it. Of 2013 cases, in which the presence or absence of a warning was noted, it was always absent in 858, or 43 per cent.; while some aura existed, at least occasionally, in 1145, or 57 per cent. Thus, roughly speaking, loss of consciousness precedes or accompanies the first symptoms in two fifths of the cases; in the other three fifths the patient is aware of the commencement of the attack. We may consider what facts regarding the modes of onset these 1145 cases supply.

FORMS OF AURA.

The sensation or motion with which the attacks commence may be referred to almost any part of the body, limbs, head, trunk, organs of special sense, and to some viscera. We must therefore infer that the process of the fit may commence in any part of the brain in which these various parts are represented. We may, for the present purpose, classify most of the auras * or modes of onset into seven groups:—(1) The unilateral auras, a motion or sensation in one side of the tongue, face, trunk, or in one arm or one leg. (2) Certain general auras; bilateral sensations in the limbs, tremors, starts, malaise, faintness, &c. (3) Auras referred to certain organs, mainly to those to which the pneumogastric nerve is distributed, and to this group belong most of the visceral warnings. The most common is the well-known epigastric sensation, and others are a feeling of choking, dyspnœa, nausea, and cardiac sensations. (4) Ver-

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* The word aura is so firmly established in medical English that it seems needless and pedantic to employ the classical plural, either the Latin *auræ* or the Greek *aurai*.

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tigo and other allied sensations. (5) Certain sensations in the head, pain, &c. (6) Psychical auras, the consciousness of an emotion or an idea. (7) Special sense warnings; some of these are strictly unilateral, but it is convenient to consider them together. This classification is clinical, and as such is adopted as affording facilities for the determination of facts apart from theories. Our knowledge of the significance of many warnings is still too imperfect to permit an exact scientific classification to be framed. It must be remembered, moreover, that the facts ascertainable certainly under-represent the actual frequency of warnings, because an aura mentioned at the moment of onset often cannot be recalled.

UNILATERAL COMMENCEMENT.—We may take first the unilateral peripheral auras, present in 17 per cent. of those with warnings.[*] Of these the commencement was in the arm in more than half (80 cases), in the leg and face in smaller and nearly equal numbers (33 and 38 cases), in the tongue in five, and in the side of the trunk in only two cases. Such unilateral commencement is common in seizures due to organic brain disease, and the organic disease causing them is usually on the surface of the brain, in the region stimulation of which causes movements in the parts first affected in the fit. Such stimulation, indeed, as Ferrier has shown, may cause convulsions beginning in the part. But from the series of cases now under consideration all those presenting symptoms suggestive of active brain disease were excluded.

Rotation of the head, or of the head and eyes, is a conspicuous feature in a large number of fits, being apparently the result of an inequality of the discharge in the two hemispheres of the brain. The head and eyes turn towards the side on which the convulsion is the more severe. It sometimes occurs before consciousness is lost, and may be the first indication of the oncoming fit. The patient may even be aware of the deviation of the eyes as well as of the head. There may be an alternation of the deviation, the head and eyes being first turned to the side opposite to that to which they are directed during the fit. Sometimes there is a double alternate deviation. In one instance the head and eyes were first turned to the right; then the head turned towards the left, the eyes slowly following; then again to the right as the convulsion came on, which was general, but greater on the right side. This initial rotation of the head sometimes proceeds to an actual rotation of the body, which will be further considered in the account of vertigo.

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[*] The unilateral commencement attracted much attention from the time of Aretæus, and Herpin (*loc. cit.*, p. 389, *et seq.*) has collected a series of instances from the writings of Aretæus, Galen, Alexander Trallianus, among the Greeks, and Ali-Rodoham, Brassavola, Sylvius, Faventinus, Dovinctus, and Hollier among the writers of the sixteenth century, of cases commencing by an aura in the hand, foot, calf, face, &c. They were also studied by Bravais in 1824, but were not, as is sometimes erroneously stated, first described by him. They have been most carefully investigated by Hughlings Jackson, to whom our present knowledge is largely due.

A curious feature of the motor commencement of a fit is sometimes presented by the cases of deviation of the head—a sense of compulsion of movement, which seems voluntary but under the influence of a sensation that the movement must be made. A patient whose fits commenced by a conscious deviation of the head, described first a sensation of discomfort in the neck and a compulsion to turn the head, which he must yield to, and he felt uncomfortable because he could not move it far enough. In another case fits began with a sense of turning to the right, manifested by deviation of the head, and attended by a subjective sensation that he must look over his right shoulder. The same sense of compulsion to turn the head was present in a patient who always tried to resist it, but in vain. In another instance a momentary sense of depression was followed by an irresistible impulse to look over the left shoulder. Then consciousness was lost in a general convulsion in which the head was directed to the left. A discrepancy in the deviation of the head and eyes was present in a case in which the eyes turned after the head; the same was conspicuous in a case in which, at the onset of right-sided fits, the head turned to the left while the eyes were directed to the right. The significance of features of deviation, especially in relation to the movement of the eyes, will be considered in connection with visual warnings. Occasionally a visual aura seems to compel a movement of the head and eyes to follow it.

Cases in which a sense of movement of the head initiates the fit rarely present the remarkable feature which is met with in the arm, a sensation of spasmodic movement without actual movement. One patient, however, described the constant warning as, first, a sensation at the tip of the nail of the great toe, which passed up to the knee and was followed by a feeling as if the head were bent violently back, so much so as to curve the spine and bring the head forwards between the legs. There was no corresponding movement, merely slight general convulsion.

TONGUE.—In five cases only the aura was referred first to the tongue; in a few the tongue was affected secondarily. The commencement may be by a movement, by a general sensation; sometimes a tingling or a feeling compared to something crawling on the tongue. Once a tingling in the tongue was accompanied by a similar sensation in the roof of the mouth; a "dreamy" feeling also attended it; vomiting always followed. In another patient the sensation of something crawling on the tongue was followed by a feeling of sickness, then of something rising in the throat, and then by palpitation of the heart. An association of sensation as well as motion in the lip and tongue was illustrated by a case in which the aura was a pricking sensation in the lip all round the mouth, which spread to the tongue on both sides, then pain was felt in the palm and consciousness was lost. In another patient a sensation of numbness round the mouth was followed by pain in the left jaw, as if the teeth were being drawn out, then loss of consciousness. In one of the cases of unilateral convulsion this was right-sided, and in each case in which the side of the tongue affected was noted, this was also the right side. Hence it seems probable that the sensori-motor processes for the tongue predominate on the left side of the brain, a fact also suggested by the great impairment of the movements of the tongue frequently noticed in right hemiplegia with aphasia.

Initial deviation of the jaw to one side sometimes accompanies a lingual aura. For instance, a tingling in the tip of the tongue spread back to its root and to the palate, and was followed by spasm of the muscles above the hyoid bone and deviation of the jaw to the left. More severe attacks began with such deviation of the jaw only, without a sensory aura. We have here a reproduction of the intimate connection between the sensory structures related to the tongue and palate and the muscles of deglutition and mastication. The association of the jaw movements with the fifth nerve may be presented by the aura of pain. In one patient pain in the left side of the head passed down to the left side of the lower jaw, and was followed by twitching of the jaw, referred to the masseter, and then by loss of consciousness and general convulsions.

FACE AND NECK.—The seizure commenced in the face in 38 cases, and on one side as often as on the other, but more frequently by a motion than by a sensation. In eight cases the mouth was drawn to one side, as if by the action of the zygomatic muscles. The spasm near the mouth was sometimes preceded by tingling. Commencement in the lips was rare. In two cases there was an initial contraction of the orbicularis palpebrarum, and in both cases this was associated with a sensation in the right hand, and the fits were right-sided.

A sensory aura in the neck is very rare, as we should expect from the slight normal importance of such impressions. In one patient, in whom the attacks had apparently resulted from an organic lesion in early life, a sensation passed from the left side of the neck down the arm to the left hand, ascended to the side of the mouth, and then passed down the side to the left leg. The subsequent convulsion was at first left-sided, but later involved the right side. Slight attacks occurred without loss of consciousness, in which the left side of the mouth and left hand twitched, but the patient always asked that the left side of the neck might be rubbed.

In most cases of organic disease in which the convulsion commenced in the face, the lesion has been found to occupy the lower part of the motor region. This, on the left side, is also connected with voluntary speech. Hence we find that inability to speak often accompanies a fit which commences in, or early involves, the right side of the face. For instance, a girl had minor seizures, which consisted in a sensation of tingling in the right arm, which passed up to the angle of the mouth, and then she became unable to speak for about five minutes. In left-handed persons the "speech-centre" is usually on the right, and not on the left side of the brain, and the association just mentioned was well exemplified by a left-handed man, who, at the age of thirty-one, became liable to fits which commenced by spasm in the left side of the face, spreading thence to the left arm, with loss of consciousness. Inability to speak preceded each attack for ten minutes, and persisted afterwards for the same time. Some of these cases are accompanied by a simultaneous sensation in the tongue. For instance, in one woman the aura of severe epileptic fits was simultaneous inability to speak and a peculiar sensation in the tongue as if this were being drawn back into the throat. She could make a noise but could not articulate. Then the head deviated to the left and consciousness was lost.

ARM.—The cases in which the attacks commenced in the arm were eighty in number. In about three quarters of the cases the commencement was in the hand. Of those beginning by spasm, twitching, &c., the first motion was, in most instances, either in the hand as a whole or in the arm as a whole. The first spasm was rarely in the fingers. In one instance there was first flexion of the thumb, then of the fingers over it, the elbow flexed, and then consciousness was lost. It sometimes begins in the shoulder, as in a case in which the attack was carefully watched, and the movement distinctly descended the arm. Often the attack begins by a sensation in the limb—"numbness," "tingling," "pins and needles," &c.; and in these the commencement is, in most, in a definite part of the hand—the forefinger (never in the thumb), in all the fingers, in the middle finger, in the palm, in the back of the hand, in the hand as a whole, and at the wrist. It seldom begins higher up the arm. In some cases the sensation with which the fits begin is less simple; it may be a distinct motor sensation. One patient always felt "as if his arm were drawing up," and would beg that it might be held down, although there was no motion in it. The feeling was accompanied by severe pain "as if the arm would break." In another there was a somewhat similar pain, "as if the arm were withering up." In one there was a feeling of twitching in the first three fingers, apparently without movement, and in a fifth there was a sensation in the back of the arm "as if the nerves were being drawn." In another patient the attacks began by twitching in the thumb and forefinger, visible to others; the arm then dropped by the side, but always seemed to the patient to be raised up over the head by spasm.

The attacks beginning with distinct spasm, or with the sensations last described, probably motor, began in the right and left hand in an equal number of cases, but those commencing with a simple sensation began in the left hand twice as frequently as in the right. A sensory aura which began as tingling, sometimes increased to actual pain as it became intense. In one case a feeling of "pins and needles" in the right forearm passed up to the face and then became general, but, in passing up the arm, it was changed, there only, to a sense of pain, as if a knife were being plunged into the arm, and this was the seat of clonic spasm which afterwards became general.

One variety of commencement in the arm deserves special mention on account of the confirmation which it affords of the distinction between the initial motor and sensory symptoms as indicative of the seat of the initial discharge in the motor or sensory structures respectively. The cases are those in which a sensory aura descends the limb, and spasm is not associated with it until the sensation reaches the extremity. For instance, a man had attacks which began thus:—"He wakes in the night with a 'rushing sensation' in the right shoulder, and this gradually, in about thirty seconds, passes down the arm to the hand, there being no spasm. When it reaches the hand, however, spasm comes on, the hand 'shuts up,' and he loses consciousness." Some cases will be mentioned presently in which the sensory aura, descending a limb, reascends, accompanied by the spasm which commences when the sensation reaches the extremity. It seems as if the resistance within the sensory centre is less than between sensory and motor centres, and that the latter is least in the part in which the extremity of the limb is represented (as may well be when we consider the extremely delicate functional relation between sensation and motion in the fingers and hand). Thus a slight discharge in the sensory centre may extend, gathering strength as it proceeds, until it reaches the part in which the extremity is represented, and then, unable to pass further, it overcomes the resistance between the sensory and motor structures, and sets up spasm in the related muscles, and the discharge passes back in the previously undischarged motor centre, so that, in this case, the sensory aura descends, the motor aura ascends the limb. But the resistance between the motor and sensory centres, even in the part in which the extremity is represented, must be greater than that within the sensory centres, because in many cases a sensory aura, beginning in the fingers, may ascend the limb without any spasm, as in some cases to be mentioned immediately.

In the majority of cases (three quarters) in which the attack begins in the hand, consciousness is lost before the seizure has extended beyond the arm. In other cases it passes to the head, trunk, or leg before the patient becomes unconscious. When to the head, it may be first felt in the side of the mouth. For instance, in one the initial symptom was a sensation of tingling, without movement, in the left hand, followed by similar tingling in the left side of the lips and nose. In another, a sensation passed up the arm to the side of the face between the mouth and nose, and was then felt in the side of the tongue. It is very rare for the aura to pass from the arm to the throat, to which the epigastric sensation so often extends. In one case in which this was described, careful questioning made it clear that the sensation was merely in the neck, not in the throat, and due to strong deviation of the head before loss of consciousness.

Occasionally the aura, passing up the arm to the shoulder, then descends the side of the trunk to the leg and to the foot. These cases will be again alluded to. It is very rare for the aura, after passing to the head, to descend the trunk; but in two cases, after ascending the arm to the mouth, it recommenced in the foot and passed *up* the leg.

Occasionally the side on which the attack begins is not uniform. In one patient the commencement was by a drawing up of one arm, sometimes the left, sometimes the right. A similar variation is occasionally met with in the part of the side first affected, as in a case in which the commencement of the fit was sometimes in the hand, sometimes in the face. In a woman aged 41, in whom fits began after a severe fright at 40, the aura was a sense of "numbness" beginning either in the hand or in the foot, sometimes in the right, but usually in the left; and in the same manner on each side. Whether it began in the hand or the foot, it ascended to the face, to the cheek and the side of the tongue, and if proceeding from the arm it afterwards passed down the side to the leg, whichever side was attacked, and she was unable to speak during the sensation. A little clonic spasm, involving the face last, followed the aura, which was prolonged, lasting a quarter of an hour or more. It was suggestive of a relation to the warning of migraine, although no headache followed.

The commencement of the fit was referred to both arms in a few cases, either by twitching or by a sensation. In one case, after tingling in the arms, these were put back and strongly abducted at the shoulder. In another, in which the aura was pain, the subsequent convulsions sometimes involved the arms only, sometimes the legs after the arms. In one case of minor epilepsy there was a peculiar sensation in both hands, which were automatically rubbed together during brief unconsciousness.

Legs.—The seizure commenced in the leg in 33 cases, and in most of these in the foot. In some it distinctly began as a movement in the toes or in the foot as a whole. In one case the movement, twitching, began in the hip, and passed down the leg.* In some cases the commencement is by a simple sensation, in the great toe, in the sole, in the dorsum, in the foot as a whole, or in the leg as a whole. A motor sensation without movement was described in one case; the patient suddenly felt as if the leg were bent up under the other, and that he must stretch it out. When the commencement is in the foot, consciousness may be lost before the aura has extended beyond the foot, but more frequently the aura extends up the leg and thigh, and it may pass up the side to the head. Occasionally, after passing up the side, instead of going to the head, it passed down the arm to the hand, and then consciousness was lost. It is very rare for an aura, after passing up to the head, to deliberately descend the arm—just as we have seen that the aura, commencing in the arm, if it ascended to the head, rarely afterwards passed down the side. The passage of the discharge to the region in which the head is represented appears to be closely connected with loss of consciousness. Sometimes the sensation, commencing in the foot, passes up the side, and when it has got to the level of the hand, it recommences there, and then passes up the arm. Conversely, in one case in which the aura commenced in the forefinger, and, after passing up to the other fingers, ascended the inner side of the arm to the face and tongue; while passing up the arm it began in the great toe and passed up the inner side of the leg as far as the knee. There are thus two modes in which the arm is involved secondarily to the leg, just as there are two modes in which the leg is involved secondarily to the arm,—one extension by continuity, passing from the one limb to the other by the trunk, and passing down the limb secondarily involved; and the other by separate commencement in the extremity of the second limb, and the passage of the aura *up* both. The facts ascertained make it probable that in the cases in which the warning passes from one to the other by continuity through the trunk, the discharge in the sensory centre takes the lead, and determines the course of the aura, although it may be closely, even instantly, followed by the motor discharge. The representation in the brain of the cutaneous nerves must be continuous as the skin in which they are distributed. The passage of the aura by continuity up the arm, down the trunk, and down the leg, or *vice versâ*, is intelligible on this theory, but scarcely on the theory that the discharge in these cases begins in the motor centres.

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* A very interesting case of this nature, in which the convulsion commenced in the groin and passed down the leg, was recorded in 1700 by Bonet (' chretum,' vol. i, sect. 12

This theory is entirely supported by the details of the cases which have come under my notice. In all cases in which an aura passed from the extremity of one limb to the other limb by the trunk, the warning was sensory in its commencement. The most striking confirmation, however, is afforded by cases (analogous to those in the arm described on p. 56) in which the warning remains purely sensory until the aura has reached the extremity of the *second* limb, and then spasm is superadded. One or two instances of this may be mentioned. In one patient the attacks always began with a sensation in the foot, without motion, and the sensation passed up the leg and side, and down the arm to the middle finger of the hand, and then only was spasm added; the hand began to twitch, and the spasm passed *up* the arm, and then consciousness was lost. In another patient the attacks begin by a sensation of "numbness" in the left wrist, which passes up the arm, down the side and leg to the foot; then it begins to ascend the limb again, and immediately the limb draws up. In a third case the attacks begin with tingling in the backs of the fingers, and this tingling passes without spasm up the arm and down the side to the leg, and then the leg draws up. A woman has minor attacks which begin with a pain in the toes of the left foot; this pain ascends the leg without any spasm, passes up the left side of the trunk to the side of the head, temple, and side of face near the nose, and next passes down the arm and into all the fingers, which feel as if they were swollen; they then begin to twitch; the arm is slightly flexed at the elbow and shoulder-joints, and then there is twitching of the face near the eye. When the pain gets into the arm she becomes unable to move it. There is no loss of consciousness, but she also has severe attacks in which she bites her tongue.

The relation between the motor and sensory phenomena in these cases is, as already suggested, to be explained by the connection between the motor and sensory centres involved, and the relative resistances in them. A discharge beginning in one functional centre, having reached the limit (representation of the periphery), may then, as already stated, not only pass to the related centre, but may return in the same centre if the discharge is slight, and has little tendency to increase in intensity. In one patient, for instance, minor attacks began with a stabbing pain in the left side of the chest, followed instantly by a "beating in the inside of the thigh, accompanied by shaking," which passed down to the foot and great toe, and then returned up the leg, still as "beat, beat," to the side of the head, and sometimes it would again return down the side to the toe. In another patient a somewhat similar "beating" sensation began in the big toe and passed up the leg and side of the trunk to the shoulder, down the arm to the fingers, and then back up the arm to the head, when consciousness was lost.

A sense of compelled movement of the leg is very rare. In one patient, in whom severe fits began in the leg, minor seizures occurred in which there was tingling in the thigh and a feeling that she must draw the leg up. Thus a deliberate discharge in the sensory centres induced a motor activity such as the will produces.

Associations of unilateral auræ with other warnings are not common. In rare cases of attacks commencing in the arm there is a subsequent special sense aura. In these the seizure in the arm begins by a sensation proper, not by spasm. This seems to indicate that, as might be expected, the special sense-centres are connected with the sensory limb-centres, rather than with the motor limb-centres. In one patient the fits began with a sensation at the elbow, which passed down to the fingers and was then felt in the corresponding side of the face and about the eye, followed by dimness of sight, and, as the sight failed, dazzling lights appeared, and then consciousness was lost. No aura *beginning* in the face, tongue, or leg was associated with one in the special senses. The only cases in which the latter was combined with a leg aura were the following:—(1) A case in which the seizures commenced with a pain in the back of the hand, and this pain, as such, passed up the arm and down the side to the leg. When it reached, still as pain, the middle of the thigh, a flash of light appeared before the eyes. The pain went on down the leg, and when it reached the foot consciousness was lost. (2) A patient, whose attacks began in the thigh, always suffered from flashes of red fire before the eyes for some days before the fit, but not at the actual onset of an attack. (3) A patient whose major attacks began with spasm in the left shoulder, descending the arm, followed by spasm in the left orbicularis palpebrarum and left side of the mouth, had also minor attacks, consisting of red and blue lights whirling before the eyes, and said to appear first "before the left eye."

In one patient, a girl, a singular association existed. The fits began with a feeling of numbness and beating in the right great toe, which passed up the leg, and when it reached the groin the patient felt a need to micturate. If she could do so, the aura ceased; if she could not, she suddenly felt as if turning over and over; then a peculiar psychical condition occurred, she lost consciousness, and passed into a severe epileptic fit. I have met with another case in which micturition sometimes arrested a commencing fit. I have also met with a case in which the act of micturition sometimes brought on a fit. The shiver, which in many children and some adults normally attends the act, indicates the associated affection of higher motor centres. One patient experienced during a few days before a severe fit, on passing water, not a shiver, but a jerk and a momentary peculiar mental state, apparently fear—the normal shiver raised to the degree of minor epilepsy. These facts, with others, suggest that the passage of urine during fits is not merely the result of loss of consciousness, but is really a part of the morbid process.

In only two cases did the attack commence in the side of the trunk; in one the sensation was described as "burning." In two cases a left-sided aura was associated with palpitation of the heart.

The mutual relation of the motor and special sense-centres in the cortex of the brain enables us to understand something of the progress of seizures. For instance, a fit beginning in the face or tongue, if it involves the limbs, takes the arm before the leg, and we find that the arm centres intervene between those for the face and tongue, near the fissure of Sylvius, and those for the leg in the upper part of the hemisphere. Again, in a fit beginning with twitching of the angle of the mouth, rotation of the head commonly succeeds, and the centre for rotation of the head is situated in front of, and contiguous to, the centre for the action of the zygomatic muscle. But in other cases it is difficult to understand the progress of the aura from the experimental facts. For this there are probably three reasons:—First, we know little of the degree of structural association or separation of these centres, the cerebral sulci, of course, affording no information on the matter; secondly, the centres represent in many cases complex movements, and fits begin usually with simple movements; thirdly, we are almost ignorant of the mutual relations of the sensory centres for the limbs, except that we know that they are intimately related to the motor centre. The facts of many auras make it probable that these, rather than the motor centres, lead in the discharge, and determine the course of the commencing fit.

A rare but curious condition sometimes attends the onset of a fit which begins unilaterally; it may be termed the alternation of the aura. In some right-sided convulsions, from left meningeal hæmorrhage, which I once watched, the rotation of the head to the right which accompanied the fit was preceded by a rotation to the left, and this itself was preceded by a slight initial rotation to the right—a double alternation. I have met with a few indications of this phenomenon in idiopathic epilepsy, but it is rare.

The great majority of fits which begin unilaterally are the result of a local cerebral lesion in early life, which having cicatrised establishes local instability, and this gradually, by repeated discharges, involves a wide extent of the brain. A unilateral onset may occur, however, in cases in which there is no evidence of a local lesion, but, on the other hand, in which an hereditary disposition affords reasonable ground for assuming the "idiopathic" form.

Bilateral and General Warnings.—In the next group are placed together the bilateral sensations in the limbs, and certain general sensations which seem to the patient to be universal. Of the attacks beginning simultaneously in the limbs of both sides, the commencement was generally in the arms. In some it was by a sensation only—numbness, tingling, or pain; more often it was by twitching, starting, jerking, or cramp in the limbs, with or without tingling. In most cases the commencement was in the hands as a whole, as might be anticipated from the wide distribution of the discharge. Tingling may be felt in both hands; then these are felt to close, and consciousness is lost. Or there may be only vague numbness in both hands. Sudden starts of the arms are an occasional form of minor attack. Attacks may also commence simultaneously in both legs, generally in the feet, as a tingling, trembling, or creeping sensation. Rarely there is a simultaneous sensation in the hands and feet.

An aura referred to the trunk, and not apparently visceral, is very rare; and the sensation is almost exclusively felt in the back. It is usually a pain or a sense of trembling or "creeping" in the spine. In one patient the sensation was that of something crawling up the middle of the back, which ascended higher and higher until it reached the back of the head, when there was giddiness, loss of sight, and then loss of consciousness, and an epileptiform convulsion. The spinal aura not uncommonly, as in this case, seems to ascend to the back of the head.

Two cases of this class afforded the only instances I have met with in which the sensation was of the character which corresponds literally to the term "aura" (see p. 46). In one the warning was described as air blowing on the skin of the epigastrium; the sensation ascended to the head, and was followed by beating of the heart and choking. The other case was a lad of 17, in whom the warning commenced with a sensation of guns going off in the head, then his sight became dim, and then he had a feeling as if wind was blowing on the skin, which commenced at both knees, and passed up to the epigastrium, and he then lost consciousness.

The other auras, grouped together as general, tremor, startings, jerkings, malaise, faintness, were described in 51 cases—one tenth of the whole. General tremor or shivering, present in 13 cases, was associated in three with pain in the head. General starts or jerkings, the aura in ten cases, were never associated with pain in the head. In several of the cases the fits occurred during sleep; the patient was awakened by the starts or jerks, and then passed into the fit. General sensations were described in 15 cases; a feeling of general illness or malaise, of general powerlessness, of general heat (in three cases), of general cold (in one case). A sense of faintness, which the patient could not localise, was the aura in 13 cases, and was associated with sweating in one case and with giddiness in three. Warnings of this class are difficult to describe, and much patience is needed to ascertain their precise character, which sometimes, after all, remains more or less uncertain.

EPIGASTRIC AURA.—The most frequent of somatic warnings is a sensation in some part of the body, usually at the epigastric region, rarely about or below the umbilicus, sometimes post-sternal, seldom to left or right of epigastrium, never to right or left of the lower part of the abdomen, sometimes to the left of the sternum, but seldom, if ever, to the right. In most cases the sensation is referred to the epigastric region, and therefore it is well to adhere to the term "epigastric" aura. But the sensation often passes to the head or the throat, and causes at the throat a sense of dyspnœa. When stationary at the epigastrium there may be nausea. This aura is referred to the functions subserved by the pneumogastric nerve.

These warnings were described in 195 cases. It is usually a vague sensation which the patient cannot precisely describe, but in a considerable number of cases it is a feeling of actual pain. It is noteworthy that, while the vague sensation often passes to the throat and head, that of pain generally remains stationary.

The character of the epigastric aura can seldom be described, partly because it is too brief to leave an effect that permits recall, sometimes because the recollection is of a sensation so unlike anything within the range of experience as to baffle all attempts to find words that convey the desired meaning. The nearest description given may be "a funny feeling." But sometimes it is a sense of movement, especially of a "turning over and over," "creeping," "winding up," "trembling," very rarely of heat or coldness.

The aura that is not definite pain generally ascends to the head or throat, very rarely to both. The feeling passes to the throat twice as frequently as to the head. Occasionally there is only a feeling that the aura passes upwards, *e. g.* to the top of the sternum. Very rarely the sensation goes to both head and throat, to the cardiac region, to the back, or to both arms. Occasionally it seems to spread over the whole body.

As a rule, when the aura reaches the head, consciousness is at once lost. It seems as though the sensory representation of the head were very closely related to the function on which consciousness depends. Occasionally the feeling passes over the head to the occiput and even to the neck, or a psychical state, such as horror, occurs before consciousness is lost.

The epigastric sensation, as already stated, often passes to the throat, and there may give rise to a sensation of choking and dyspnœa. This reminds us of the well-known sensation in hysteria, and in many cases of genuine epilepsy the sensation appears to be exactly the same as the globus hystericus.* Not only so, but I have seen a case of tumour of the pons varolii in which epileptiform convulsions commenced with the same sensation, described as a "ball rising up from the stomach to the throat, and stopping the breath." The sense of choking is sometimes very intense. One patient described it as if the palate and larynx were pressed together.

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* The close association of the globus hystericus and epilepsy attracted the attention of Dr. Parry. ('Medical Writings,' London. 1825, p. 392.)

Just as the warning commences sometimes to the left of the epigastrium, but never to the right, so when it is felt as ascending through the chest on one side, this is always on the left side, never on the right.

These auras felt in or ascending to the chest and throat appear to be the expression of a disturbance in the cerebral processes connected with the respiratory function of the pneumogastric. Spasmodic actions form part of the normal action of the respiratory centre as they do of no other, and must be reproduced in higher and associated centres. The epigastric pain which remains limited to the epigastrium appears to be related exclusively to the gastric division of the vagus. Accordingly we find that there was usually described, as part of the aura, another sensation referable to the same function—nausea, evidently such after careful questioning, and accompanied in several cases by actual retching. In one patient, the feeling in minor attacks always made him take his handkerchief out, expecting to be sick, but as soon as it was ready the sensation was gone. In this case there was smacking of the lips, but no taste was remembered; even consciousness was preserved throughout. With this may be associated another curious case in which the initial nausea caused vomiting, but after some time the vomiting was replaced by spitting. In another case sudden, intense hunger was described. Other symptoms, often associated with gastric disturbance, accompanied the pneumogastric aura in some cases, as pain in the head and giddiness, the latter especially when there was actual nausea, the former when there was actual pain in the epigastrium.

The accompanying vertigo is an instance of the association so common in every form of giddiness. The feeling has been described as identical with sea-sickness. A dreamy sensation sometimes attends nausea and giddiness.

A sudden sense of gastric distension is sometimes felt, with or without nausea, and may be accompanied by actual eructation. The attack may pass off with the flatus, and come on if no flatus can be expelled. We are not justified in assuming from this that flatulence is the cause of the attack, any more than we are in regarding gastric disturbance as the cause of an attack of migraine which subsides with vomiting.

CARDIAC SENSATIONS.—Another organ within the innervation of the vagus is the heart, and cardiac sensations constituted the aura in a small proportion of cases,—pain, a vague sensation, or, much more commonly, palpitation. In one patient the attacks were usually preceded by an epigastric sensation passing up the left side of the chest to the head, but in some attacks this was preceded and followed by severe palpitation of the heart and dyspnœa. Cardiac palpitation and discomfort are sometimes associated in an aura with "tingling" in one or both arms.

VERTIGO.

In no less than 18 per cent. of the warnings the aura was sudden vertigo, generally definite. The attacks commenced with a sense of giddiness, vague or definitively vertiginous, or with the fact of rotation. As a rule, when a sensation merely, the direction cannot be ascertained, but sometimes there is a distinct sense of turning to right or left, or of a corresponding movement in objects. Of 15 cases with actual rotation, the movement was to the right in 5, and to the left in 10. In nearly all the convulsion was bilateral. In bilateral fits the discharge is never quite equal on the two sides, as proved by the common deviation of the head, and the vertigo, when slight, may be the expression of this inequality. In most consciousness was early lost.

As already stated, giddiness is often associated with an epigastric sensation, especially when this is distinct nausea. It may also be associated with loss of sight. In one patient, for instance, objects before him suddenly appeared to turn round and round, and, while doing so, gradually became indistinct, and he lost consciousness.

CEPHALIC AURAS.

Sensations referred to the head preceded the fits in 90 cases. This warning is of interest, because a cephalic sensation seems often to be associated with a mental state; when a sensory aura begins in the limbs or epigastrium and ascends, consciousness is usually lost when it reaches the head. It is not, therefore, surprising that a primary head sensation should be, as a rule, followed directly by loss of consciousness.

In about a third of the cases the sensation was actual pain, general or local, frontal, vertical, seldom occipital or felt through one side of the head, but, in a few, referred to one temple. Local acute pain may simulate neuralgia, and may mislead, if the patient is unaware of the loss of consciousness. In one the warning was violent pain through each zygoma, and after the fit there was local tenderness at the seat of the pain. Cephalic pain may be attended by vertigo, usually vague, by nausea, or by somnolence.

In 55 cases (about two thirds) the cephalic sensation is other than distinct pain.

A dim sense of heaviness or pressure or fulness may be experienced, sometimes with somnolence, or a sense of strangeness. A feeling that is not uncommon is a sudden sense of "rushing" in the head, sometimes described as "blood rushing" to it from the trunk or the limbs.

Often this aura is so peculiar as to be quite beyond description, save by some metaphorical expression which conveys but little meaning, such as "numbness inside the head," "faintness going over the brain," or "something stirring the brains round." The last, which followed the epigastric throat aura, is suggestive of vertigo, especially in connection with other cases of what can only be termed "intra-cranial vertigo." In one patient, for instance, the sensation was described as "two circles within the head moving rapidly in opposite directions, and seeming to get alternately nearer and farther off," until consciousness was lost.

A visual aura is scarcely ever met with in association with a cephalic sensation, but a few cases presented also an auditory aura, of simple character, and of special interest in connection with sensations in the head allied to a sense of sound. In one case an epigastric sensation seemed to ascend the left side of the head to the left occ [illegible] there to terminate in a "bang," which was constantly averred to be a physical sensation, and not a noise.

It has been said that a trunk or limb sensation, which ascends, when it reaches the head is associated with loss of consciousness. Hence the mental states that are sometimes, although rarely, associated with a cephalic aura are of special interest.

PSYCHICAL AURAS.

The aura was psychical in 92 cases,—a peculiar mental state, remembered afterwards sometimes in definite detail, sometimes only in vague character. Extreme care is necessary in investigation if accurate facts are to be obtained. A psychical aura is especially common in minor attacks, and their occurrence can only be ascertained by asking for their chief forms; such information has afterwards to be weighed and sifted by cross-examination, because the occurrence of attacks of which they may be the only evidence is often unsuspected.

In about a third of the cases the warning of the fit was an emotional condition, generally that of fear, sometimes described as a feeling of "horror." In only one instance was this aura related to fright as the exciting cause of the first fit. The feeling was generally vague, without any association with an idea; but in some cases there was a definite mental conception; *e. g.* that a man was behind the patient from whom escape was necessary. In one case the fear involved the idea that the man was about to jump on the patient's back.

In one case, such a mental state seemed to be the cause of "cursive" epilepsy. The patient generally ran, with an alarmed expression, looking back and saying "It is coming." Yet the mental state could never afterwards be recollected. Hence a similar emotion may be the cause of the run in other cases in which neither the movement nor emotion are remembered. In one case, a girl aged 13, the sense of fear was invariable, but only sometimes did it cause her to run; occasionally, as if the cause were in front of her and not behind, she put her back against a wall, with a look of fright. An epigastric sensation sometimes preceded the emotion in minor attacks but not, as a rule, in the severe seizures.

This is quite different from, and cannot be confounded with, the natural feeling of alarm when the first indications of a coming attack are relalised. If these exist, they are always definite, and quite obscure any emotion they may cause.

Attention has been called to the association of the aura with the epigastric sensation, but it is not a frequent combination. In one patient an epigastric sensation, or red stars, preceded the manifestation of extreme terror, not afterwards remembered. A throat aura might be expected to be felt, since fear so often normally causes a sense of choking, but the association occurs rarely, if ever, perhaps because the emotion passes too directly into loss of consciousness. Occasional, but also rare, are associated vertigo (which may be objective or subjective), a visual or an auditory sensation. In one patient the sound of bells was accompanied by intense fear, a connection for which it is not easy to conceive a physiological reason.

Allied to fear, but far less common, is a sense of intense misery, as if some "bad news" had just been heard. One patient suddenly saw stars, and then felt intensely depressed "as if at a funeral."

Equally rare, but perhaps more curious, is a sudden sense of personal wrong-doing. It may have the tone of a dread of being found out. Once it occurred when an ascending epigastric aura reached the head, and once it was followed by sudden nausea, retching, and vertigo.

This sense of wrong takes us (perhaps, however, not by a natural sequence) to the sense of strangeness and unfamiliarity which is an occasional aura. It is common as the subjective effect of the onset of minor epilepsy, and it is occasionally the warning of a severe attack. It is more frequent than a sense of fear. There is a sudden impression that the surroundings are unfamiliar. The place and persons appear strange; objects seem not to have been seen before. Our consciousness of the familiarity of surroundings depends, of course, on the mysterious recognition of the correspondence between the present visual sensations and previous ones, recognised by their residual effect. The residual disposition to renewal, which is the basis of memory, must always facilitate perfect perception of surroundings, and this ease of perception we feel as "familiarity." It is possible that a sudden simple difficulty in perception, not enough to cause dimness of sight, may have an effect opposite to that of ease, and, as the latter causes a place to seem familiar, its sudden absence, the increased effort to perceive, may by itself result in a sense of strangeness. One patient described the feeling as "suddenly seeming to be somewhere else."

Another sensation sometimes perceived may be merely an imperfect degree of that just described, but it is interesting as resembling that which sometimes occurs in health, as will be remembered by readers of the "Autocrat of the Breakfast Table." It is the sensation of that which is happening having been previously experienced in the same form. This may seem the converse of that just described, but in one patient the aura was a sudden feeling that the surroundings were strange and yet had been familiar long ago and had been forgotten. Sometimes it amounted to a sudden recognition of the place as that in which the whole previous life had been spent. Tempting as the subject is, the further analysis of this aura would carry us too far.

This sudden sense of unreality passes into a peculiar "dreamy state" which is a not unfrequent feature of minor attacks. It apparently involves a greater change in consciousness, so that sensory perception is less acute and may cause an apparent distance of sounds. It may be associated with movement of the lips, as if some taste were perceived (to which Hughlings Jackson has called attention).* In one instance a sense of unreality was attended by an olfactory aura, and occasionally the epigastric aura precedes it.

From such conditions in which mental activity is diminished, rare cases present various transitional forms connecting these with positive mental activity. These sometimes involve psychical sensations, the definiteness of which is often obscured by imperfect subsequent recollection. In one patient a sentence always seemed to come into his mind, of which he could only now and then remember that it seemed to be some line of Virgil which he had learned at school. In another there was an impression of seeing distinctly events of past life, although the patient could never recall them so as to say what they were. A sudden intense desire to be alone is sometimes described. Another of the infinitely various states that occur was a vague but strong feeling that something was wanted, and that an effort must be made to obtain it, but the nature of the want could never be recalled. One patient woke up with a sensation of having a vivid dream and then became unconscious in the fit.

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* In one case of the kind a small spot of softening was found in the uncinate gyrus (Hughlings Jackson and Colman, 'Brain,' 1899).

More pronounced is a consciousness of ideas suddenly rushing through the brain with uncontrollable energy; in one case the patient woke from sleep with a vague sense of mental energy and then passed into the fit. But such indications of pure ideational energy are rare, and present a very small proportion to the cases with some degree of psychical inhibition or perversion. These psychical auras are often scarcely separable from the higher special sense warnings. It is obvious that the distinct idea of a sentence and perception of its sound may be almost identical in significance.

SPECIAL SENSE AURAS.

The last group of auras are those referred to the special senses and their organs, which were present in 274 cases. Two thirds of these (175) were visual, one fifth (68) were auditory, 12 olfactory, and 18 were apparently gustatory. The great difficulty of distinction of many gustatory auræ will be mentioned presently.

Olfactory.—Sensations of smell preceded the fit in 12 cases, all purely epileptic. In most cases the sensation was of an unpleasant smell. One patient described it as a smell like "matter"; others compared it to gas and sulphur. Occasionally the smell seems to come from the stomach. Exceptional comparisons were to Tonquin beans, to carbolic acid, and to magnolia. It was described in these cases as a definite smell, and not as a flavour.

A flavour is an olfactory sensation produced by that which reaches the nerve endings through the posterior nares, and may blend with a true gustatory sensation. In some cases the aura was clearly a "flavour," *i. e.* an olfactory "taste," not a distinct smell. Thus, auditory and visual sensations (sound of bells with red and blue lights) were followed by a nasty taste, as if "everything was going bad." In another aura the sensation was described as the taste of a cherry. One warning was described as both a smell and a taste of bad gas, the taste being evidently the olfactory "flavour." It persisted longer as a sensation of smell than as a "taste." This comparison to the smell of a gas of some kind is not unfrequent, and is sometimes so strong that it seems to the patient to pervade the head and even the whole body.

The chief associations noted were a visual aura, and also a visual aura succeeded by a sensation of bells ringing.

The distinct differentiation of smell and "taste," used in the sense of flavour, is important. Care was taken in each case to ascertain the character of the sensation, and it is certain that the olfactory "taste" is always uniform in the same patient, and so is the olfactory "smell." The latter is probably sometimes blended with a true gustatory "taste," as a sensation of bitter accompanying one of smell. But the common distinction indicates that the central relations of the two olfactory functions are disjoined. A sensation of a smell was always distinguished from one of a flavour. A nasal warning was present in a few cases, all epileptic, and consisted in sudden tingling in the nose.

Gustatory.—A true gustatory aura is very rare, occurring perhaps once in two hundred cases. It was variously described—as a taste "between sour and bitter," referred to the back of the tongue; or as "between bitter and sulphur." In others it could not be described, but was referred to the mouth and in some caused a tendency to swallow. In a patient with post-hemiplegic convulsions, the aura was a metallic taste in the mouth, "like copper," with a simultaneous painful sensation in the thumb and forefinger of the left hand, which passed to the other fingers, and then up the arm to the side of the face, and down the body to the leg, and "seemed to go out at the great toe." In one patient the aura seemed to bear the same relation to taste as tingling in the nose to smell, or pain in the eye to a visual aura. But a discharge in the brain involving the taste-region may be more frequent, because it is not uncommon, especially in minor attacks, for smacking of the lips, and even moistening them with the tongue, to suggest to an observer that some sensation of taste is the cause of the movement. It may, however, also be due to an (olfactory) flavour; indeed, the patient whose minor aura was the smell "magnolia" always moved his lips as if tasting. Dr. Hughlings Jackson has observed this to be associated with a "dreamy" sensation (see p. 71), but the association occurred in only one of the cases now analysed. With the exception of the cases just mentioned, no associations of the gustatory aura were observed.

Ocular and Visual.—The warnings referred to the organ of vision may be divided into five classes: a sensation in the eyeball itself; diplopia; an apparent increase or diminution in the size of objects; loss of sight; distinct visual sensations. A sensation in the eyeball was present in seven cases, five being purely epileptic. The eye to which it was referred was in one case on the same side as the first motor symptom of the fit; in one on the opposite side. It is probable that the centre for common sensation in the eyeball is connected with that for vision. This is suggested by the photophobia due to irritation of the fifth nerve,—the way in which light causes actual pain in an eye the seat of superficial inflammation. In one patient, fits commencing with a pain in the eye were followed by red vision; and in a case of cerebral tumour presently to be mentioned, a visual sensation and pain in one eye were associated as the aura of the fits. Double vision preceded the attacks in a few cases. In one the diplopia was associated with aching in one eye and an apparent magnification of objects, which looked "twice their real size."

The apparent increase or diminution in the size of objects is a rare and curious aura. It is presumably connected with increase or diminution in the sensitiveness of the visual centre, which produces the same effect on consciousness as the stimulation of a larger or smaller area of the retina by a larger or smaller image. It is therefore not surprising to find that diminution may be followed by loss of sight. For instance, in one case objects appeared to recede, becoming smaller and less distinct. If the patient was going to a door, for instance, she would have to feel for it, because it appeared small and dim; the indistinctness increased until sight was almost gone, and then consciousness was lost. Loss of sight preceded loss of consciousness in 26 cases. It was commonly complete and simultaneous in the two eyes. In one case the sight of the right eye went before that of the left, and this was followed by a rotation of the head to the left. In one case the loss of sight in minor attacks lasted for an hour, and was followed by a sleep for an hour. The loss was preceded by a visual sensation in two cases, by an auditory sensation in one, and in some cases loss of hearing accompanied loss of sight. The only frequent associations were with vertigo in four cases, and with rotation of the head in two.

The aura is often a distinct visual sensation. It may be that of light, not colour—sparks, a ball of light, a flash, or a glare, but often colours are seen. When only one colour is seen it is usually either red or blue—the former is more common. Green is seen alone rarely, and only as a diffused colour. Combinations described were—red and blue; red, blue, and purple; red, blue, and green; red and green; blue, green, and yellow; red, blue, and yellow. Thus not only were red and blue the only colours seen alone, but they were both present in half the cases in which other colours were seen, and in no case were both absent. Taking the cases together, the order of frequency of the several colours is—red, blue, green, yellow, and purple. It is evident that this order does not correspond to the physical relations of colour, as shown, for instance, by their grouping in the spectrum; nor does it correspond in any way to the areas of retinal perception. Red, seen most frequently, has one of the smallest retinal fields; but green, seen much less frequently, has a still smaller field; while the field for yellow, seen as rarely as green, is one of the largest, and is nearly the same as the field for blue, which is one of the colours mentioned most frequently. But the order does correspond, not exactly, but in a very suggestive manner, to the degree of visibility of colours. Cohn* found that the order in which spots of colour, one millimètre in diameter, can be seen in direct sunlight at different distances, is—beginning with that which can be recognised at the greatest distance—red, next blue and green (equal), next yellow, and least violet. This is evidently not far from the order of development in epilepsy.

The apparent diminution in the size of objects, which must be referred to an inhibition of the visual centre, has been mentioned in connection with the loss of sight which it often precedes. Partial inhibition is equivalent to a diminution in the sensitiveness of the centre,—an object appears smaller or farther off; and as the inhibition of the centre increases, the object may appear to recede. It is the opposite process to that which occurs in augmented action of the centre; in this there is increased sensitiveness, and objects appear larger. But such increased activity more often results in such spontaneous discharge as causes a definite sensation. There is a visual illusion, perhaps a sense of a ball of fire; as the discharge spreads, this seems to get larger and nearer. The effect is the same as if an increasingly large area of the retina were stimulated, and similar to the slighter degree of overaction in which a seen object seems to increase in size. Thus in one case the warning was always a blue star, which appeared to be opposite the left eye, and to come nearer until consciousness was lost. Another patient always saw an object, not described as light, before the left eye, whirling round and round. It seemed to come nearer and nearer, describing larger circles as it approached, until he fell unconscious. The phenomena of some of these auras suggest that there may be both a discharge and an inhibition of the same centre, as the result of a different degree of the same morbid function. For instance, one patient always first saw a flash of light, then sight was lost, and then consciousness. So the light or other object which appears to the patient, instead of becoming larger, may seem smaller and more distant, apparently in consequence of the inhibition overcoming the discharge. Thus in one case the warning commenced by dimness of sight, and then a light appeared, whirling round and round. This appeared to get farther and farther off, until consciousness was lost. There may be inhibition of the centre in severe attacks, and a discharge in the minor seizures. Thus one patient always saw colours, especially green and blue, in slight attacks, while the severe fits were preceded by simple loss of sight.*

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* 'Brit. Med. Journal,' October 4th, 1879.

In some cases the warning is a highly specialised visual sensation, a complex visual conception, or psycho-visual aura. This may succeed a simple sensation. One patient, in attacks of *petit mal*, first saw a number of red and white dazzling stars, and then an old woman, with other objects which she could not remember. Another patient first saw always bright lights—red, green, yellow—and then, the lights continuing, she saw a girl, and "tried to get to her to ask what the lights meant," and then fell in the fit. In the first of these cases the less specialised preceded the more specialised sensation, a sequence to which Hughlings Jackson has drawn attention, as suggesting that the more elaborate sensation is the result of the loss of control from the discharge causing the less elaborate sensation. But in the second, the more and the less elaborate sensations occurred together, and in other cases an equally special sensation, a visual idea, constituted the sole warning of the fit, and must be ascribed to the discharge itself. Two patients had visions of an old woman; in one case, to be mentioned in detail presently, the old woman always had a brown dress; in three, ugly faces were seen; in two "various people," and in one "animals." Two patients described an aura consisting of visions of objects and places familiar long ago, a visual sensation analogous to the psychical aura I have mentioned. In one patient the fits were preceded by a vision of "beautiful places, large rooms," &c.

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* See the writer's Bowman Lecture on "Subjective Visual Sensations," 'Trans. Ophthal. Soc.,' vol. xv, 1895, for a more elaborate discussion of these symptoms.

Associations of the visual aura are comparatively rare. There may be a sensation in the limbs. One instance was a case in which a flash was seen as soon as a pain, which commenced in the arm, reached the middle of the thigh. When the visual sensation is unilateral, the side may be the same as that on which the limbs are involved. Thus, in one case, some fits commenced with the appearance of a blue star to the left, which grew larger and larger until consciousness was lost, while other fits began in the left arm, and some in the left leg. In another patient a bright light, referred to the right eye, was associated with loss of speech. Occasionally a visual aura is associated with initial rotation, of which the patient is aware. Thus, in one, a blue star always appeared and seemed to move to the left, and the patient felt obliged to follow it with his head; the subsequent fit was apparently bilateral. Epigastric sensations are occasionally associated, and so is an auditory sensation. The sensations in one patient were very complex and numerous, and, as I shall have to refer to it again, it may be as well to relate the case in some detail. The patient was an intelligent man, twenty-six years of age, and all his attacks began in the same manner. First there was a sensation in the left hypochondriac region "like pain with a cramp;" then, this sensation continuing, a kind of lump seemed to pass up the left side of the chest, with a "thump, thump," and when it reached the upper part of the chest it became a "knocking," which was heard as well as felt. The sensation rose up to the left ear, and was then like the "hissing of a railway engine," and this seemed to "work over his head." Then he suddenly and invariably saw before him an old woman in a brown-stuff dress, who offered him something which had the smell of Tonquin beans. The old woman then disappeared, and two great lights came before him—round lights, side by side, which got nearer and nearer with a jerking motion. When the lights appeared the hissing noise ceased, and he felt a choking sensation in the throat, and lost consciousness in the fit, which, from the description, was undoubtedly epileptic. He had also attacks of *petit mal*, which consisted of a vision of a dull-red ball to the right, in the lower part of the field. Thus we have first a pneumogastric (visceral) sensation which becomes intermitting and sound-like as it ascends towards the ear, until it is finally an intermitting hissing, perhaps the least specialised of all the auditory sensations. This is accompanied by a visual sensation of very high specialisation—a visual idea—associated with an olfactory sensation, and it gives place to a much less elaborate sensation, the two lights, and, as the visual centre becomes invaded by this discharge, that in the auditory centre ceases. Finally, the aura terminates with the completion of the pneumogastric sensation—a sense of choking. The case will be referred to again in the chapter on "Pathology," but I may here point out that the more specialised visual sensation preceded the less special, and therefore cannot have been due to "loss of control" by preceding discharge; it was evidently due to the discharge itself. The phenomenon of two lights before the eyes is one of great rarity among the auras of epilepsy. Lastly, the association of intermission of the sound with a jerking movement of the lights is worth remark. In one other patient a visual aura was accompanied by a disagreeable undescribable "smell" after it had lasted a short time, and just before consciousness was lost.

The only autopsy which I believe has been published of a case in which the visual warning existed, is one which I have recorded.* The fits were preceded by a flash of light or pain in the eye, and micropsy, and were due to a tumour in the occipital lobe, which had extended forwards as far as the angular convolution.

Auditory.—An auditory warning was described in 7 per cent. of those with warnings. In some cases there was an auditory loss, a sudden sense of stillness. This is relatively less common than the analogous loss of sight, but the two symptoms may be associated—for a moment all is silent, then all becomes dark, then consciousness is lost. In a case in which the sensation began in the tongue and passed to the arm and leg, sight was lost after the arm was involved, and hearing after the sensation was felt in the leg. In rare cases the loss succeeds an auditory sensation. In one a sound, as of a machine, was followed by deafness. In the other a "rushing," not loud, was accompanied by some visual sensation which could never be recalled, and was followed by a sense of fright, such as to make the patient burst out into a perspiration; at the same time the sounds become fainter and more and more distant, until consciousness was lost.

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* "Cases of Cerebral Tumour illustrating Diagnosis and Localisation," 'Lancet,' 1879, vol. i.

This increasing faintness of sounds that are heard, suggesting greater distance of their cause, is analogous to the micropsy already mentioned. Diminution of hearing may also coincide with, and seem to be the result of, an auditory sensation, as in a case in which a hissing sound, increasing, and culminating in loss of consciousness, seemed to prevent the patient hearing that which was said.

Of peculiar interest are rare cases in which mind-deafness is part of the aura. In one case (after mumps), a peculiar buzzing in the left ear, which was the aura of several fits, sometimes occurred alone as a form of *petit mal.* It was often excited by sound, as the noise of an omnibus or the note of a piano, and with it all sense of tone was lost. The loudness or softness of the sounds were recognised, but all seemed alike in tone. In another case a buzzing sound, the aura of severe fits with similar deafness, was always induced by a certain note of the organ, the open diapason A of the pedals.

Among the various auditory sensations which occasionally herald fits are a sensation of an explosion or crash (an aura noted by Aretæus), the "sound of a drum," a "whiz," a "hissing," a "ringing," a "rustling," a "rushing sound," and a "sound of thunder," or a "whistle." In some cases the sound was at first distant, and seemed to get louder and near, until consciousness was lost—a phenomenon analogous to that just described in connection with the visual aura.

Unilateral limb warnings, suggestive of an old organic lesion, are rarely associated with an auditory aura. In one case the commencement was in the hand on the side to which the sound was referred and the convulsion was confined. In another the sudden sound of a bell in the right ear was followed by cramp in the right leg and loss of consciousness. In a third some attacks were preceded by an auditory sensation, "singing in the ears," and others by an auditory loss—sounds suddenly became distant and indistinct.

The aura is sometimes a more elaborate auditory perception. One patient always heard the words "c-o-n with a con" repeated several times, and then came the fit. In some cases music was heard. One of these cases illustrates the fact that the slighter the discharge, the more elaborate is the sensation. Slight fits were preceded by the sound of music, a confused "air," of which the patient could never succeed in making out the tune. Before severe attacks, however, he heard only a bell ringing. An elaborate aura often takes the form of voices, but, as a rule, these more elaborate auditory sensations leave only a slight trace that cannot be recollected. In one instance an epigastric sensation was followed by an impression that a certain person always said something which could never be recalled. A similar vague impression may go so far as a recollection of definite voices, without memory of what was said.

The most frequent associations of the auditory aura are with a visual aura, and an olfactory aura. Other combinations noted were with sensations in both legs, in both arms, vertigo, an epigastric sensation, and nausea. In several cases in which a visual and auditory aura were combined, the two corresponded in character, *i. e.* in their degree of elaboration. For instance, one patient described a red light and a rushing sensation like water. The patient who heard the words "c-o-n with a con" saw a girl standing by her who was saying them; and a patient who saw "beautiful places, large rooms, etc.," heard at the same time "beautiful music." An auditory sensation may, however, be accompanied by dimness of sight, as in a case in which these symptoms were accompanied by a sensation of wind blowing upon the skin.

In connection with the association of the visual and auditory warnings we may note the contiguity of their centres. The angular gyrus, in which is probably the higher visual centre, is continuous with the superior temporo-sphenoidal convolution, in which is the auditory centre. At the same time local contiguity must not be over-estimated. Distant parts may have a close functional relation.

Visual and auditory sensations are not only associated in the same warning; they may occur in the same patient as the warnings of fits of different kinds. In one patient, for instance, each minor attack began with a visual sensation; each severe attack with an auditory aura, accompanied with vertigo.

A remarkable change of auditory to physical sensation occurred in the case mentioned, in which buzzing abolished perception of tone. The buzzing changed to left-sided hissing (the right ear being deaf), and this, each time, seemed to become a sense of vibration which passed down the left side and limbs. The fact that the vibrations in low notes can be felt as well as heard, prepares us for a relation of the centres by which a discharge in one may pass to the other. It is one of many facts which show the relation of each auditory nerve to the cortex of the opposite hemisphere, and of auditory sensations—the physical cause of which is only a little higher than touch—to lower sensory impressions. Vibrations perceived only by touch, when more frequent, are perceived by the ear. Low tones, indeed, can be felt long after they cease to be audible. It is often difficult for a patient to say whether an auditory aura is a sensation or a sound.

But the locality to which auditory sensations are referred is sometimes anomalous. They may seem to be in the back, or top of the head, or around it. Changing to a vibration, the sensation may not only pass down one side, but may seem to pervade the whole frame to the feet.

Vertigo is a not unfrequent association, as might be expected. The peripheral association of the sources of sensations of sound and those that influence the centre for equilibrium, seems repeated in the central structures. Balance is maintained chiefly by the legs; their movement in locomotion is rhythmical, and the auditory nerve is the chief channel by which rhythm is impressed on the nervous functions. Hence it is intelligible that vertigo should be sometimes associated with discharge in the auditory centre by the relation of the structures concerned.

Probably a still more important relation is due to the central connection which causes the instant turning of the head and body towards the side on which a sound is heard. It explains the readiness with which the motor centres are disturbed, for discharge in the auditory centres doubtless acts on the motor structures as readily as if it were due to an aural impression. The manner in which the blending of the allied sensations affects consciousness is sometimes illustrated in epilepsy, although generally in a manner too complex to permit more than a general recognition.

The process of such an onset is sometimes conspicuous. In one case of certainly idiopathic epilepsy (although the convulsion was greater on the right side) the aura began with a sound in the right ear; this was followed immediately by a sensation of turning the eyes and head to the right, and also of falling, and immediately after by actual deviation of head and eyes to the right. Probably a special instability of the left auditory and motor centres determined the initial discharge, but the features illustrate the effect of the sensory on the motor centre. They also illustrate the fact already mentioned that vertigo is essentially a motor sensation.

The complex relation of vertigo is exemplified by the relation of what may be called encephalic vertigo to hearing. As the aura may be a sensation of a wheel turning in the head, so the warning in another case may be described as the sound of whirling in the head—a sound, not a simple sensation. The auditory nature of this aura was illustrated by the fact that in one case, instead of that described, there was sometimes a sound as of a band playing, which became louder and louder until it "seemed to take away the senses."

CHAPTER III.

SYMPTOMS CONTINUED.

SEVERE ATTACKS.

THE symptoms of the developed attack, briefly described on p. 38, may now be considered in detail.

Epileptic Cry.—The onset of some attacks is attended by a well-known scream, the "epileptic cry." It is more frequently absent than present. Its characters vary. The true epileptic cry is a wild, harsh, screaming sound, probably due to the tonic spasm of the thoracic and abdominal muscles expelling the air through the glottis, itself narrowed by spasm. The sound has been compared by Reynolds to the "cry of a distracted peacock." It is, indeed, scarcely imitable except by the larynx of a bird. There was once a parrot at the country branch of the National Hospital, which had learned to imitate the cry so closely as frequently to deceive the nurses, who ran to the spot, expecting to find a patient in a fit. In many cases the cry is not of this character, but is a simple scream, sometimes repeated once or twice, as the fit is coming on. No recollection of the cry is, as a rule, retained. In rare cases the patient is conscious of the scream, although he is unable to prevent it.

Loss of consciousness is invariable in all severe epileptic fits. It is only in slight seizures, minor epilepsy, that consciousness may be merely impaired, or even retained. In many instances the loss of consciousness occurs at the very onset of the fit, before, or at the same time as, the earliest convulsive symptom, so that the patient knows nothing of the onset. In other cases, as already detailed, consciousness may be retained until after some other symptom has occurred, of which the patient is aware as the warning of the fit, and, in the description of these warnings, their relation to the loss of consciousness occurs as has been described. Consciousness is lost before, or at the time, the patient falls. The loss usually occurs later in cases of convulsion from organic brain disease than in idiopathic epilepsy. In the latter, consciousness is seldom preserved after the initial period of the aura. In severe attacks it rarely persists so long as in a patient who was waked from sleep by the commencing spasm, the hands clenched and jerking, with the head turned to the right. He tried to get it back, but could not, and tried to speak, but was unable to do so; then he lost consciousness.

CONVULSION.—*Tonic Spasm.*—As a rule, the convulsion of a severe epileptic fit begins by tonic spasm, causing violent, strained distortion of head, face, and limbs.

The first evidence of the commencing spasm is, in a large number of cases, a deviation of the head and eyes to one side, or an actual rotation of the head. Sometimes the head is turned "as far as it will go," without any rotation of the trunk. Occasionally the trunk is rotated, and the patient may even turn round once, twice, or three times before falling. Not only may there be this rotation before the fall, but it may (very rarely) go on afterwards, the patient rolling over and over on the floor. The eyes deviate before the head, and usually to a greater degree. They are directed towards the side to which the patient turns. This deviation is probably the result of the discharge being greater on one side than on the other. This is shown in certain severe fits in which a transient post-epileptic phenomenon (foot-clonus) succeeds the fit. If there is initial rotation of the head, the foot-clonus may be found only on the side towards which the head had been rotated. It is shown also by the phenomena of fits which are unilateral or begin unilaterally; the head and eyes are first turned towards the side on which the convulsion commences. The head is turned chiefly by the sterno-mastoid, which acts in physiological association with the muscles of the limbs on the opposite side, turning the head normally towards the arm which is in use.* The association is of the movements, not necessarily of the muscles. In a right-sided epileptic fit the spasm involves the *left* sterno-mastoid, turning the head to the right. The lateral muscles of the eyeballs, acting together, turn both eyes towards the side involved. If the spasm, after affecting the limbs on one side, passes over to the limbs on the other, lessening on the side first affected, the head and eyes are turned towards the second side when this is involved. The condition in unilateral convulsion (as Hughlings Jackson has pointed out) is always the opposite to that in unilateral paralysis from disease of the cerebral hemisphere. In the early stage of hemiplegia the head and eyes deviate *from* the paralysed side in consequence of the weakness of the muscles which should turn them to that side. Thus they deviate towards the side of the cerebral lesion. In the unilateral convulsion there is deviation *from* the side of the brain affected, and *towards* the limbs which are convulsed.

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* This physiological association determines the pathological associations of the action of the muscles; as, for example, in torticollis. In cases of that disease in which the spasm involves first the sterno-mastoid and afterwards one arm, it is the arm on the opposite side which is involved.

An alternation of the initial rotation of the head is sometimes met with. In one case, with general convulsion, the head was first turned to the left and then to the right. Still more rare is a movement of the head and eyes in opposite directions. In one patient, bilateral convulsions began with a movement of the head to the right and of the eyes to the left. In another, the head first turned to the left and the eyes to the right, then the head slowly followed the eyes and deviated strongly to the right. This is perhaps a compensatory movement, the initial tendency to rotation being too slight to involve the eyes as well as the head. The initial deviation of the head and eyes may be present in some attacks, and absent in others, in the same patient.

Sometimes a curious irregularity of the initial movement of the eyes is observed in cases of idiopathic epilepsy, but is more common in cases in which the convulsions result from an organic lesion. Thus in a case in which the definite convulsion began at the left side of the mouth, the head and eyes first turned to the left, then the head to the right, while the eyes were still directed to the left, but slowly followed the head to the right as far as the middle line, and then diverged strongly to the left. A series of nystagmic jerks then occurred towards the right, and they were finally fixed in that direction, with the head.

Since the deviation of the head must be regarded as the result of unilateral excess of the "discharge," and may progress to actual turning, it would be expected that the subsequent fall would be towards that side. But often the patient falls to the other side. This may be owing to a complementary counteraction of the tendency to rotation, carried to excess. In a child of eight, whose attacks presented an example of the slighter degree of convulsion, there was, without warning, a deviation of the mouth and head to the right, and elevation of the right arm. Then she fell to the left so constantly that the state of the left arm could never be observed. There was slight clonic spasm in the right face and arm, brief unconsciousness, and then after a few minutes' "fumbling" the attack was over. The case was apparently idiopathic, and the initial tonic spasm and slightness of the clonic spasm afford a characteristic contrast to the slight attacks due to "organic epilepsy," in which clonic spasm occurs first, and is often alone present.

When there is not merely deviation but an extreme movement of the head or a rotation of the trunk, there may be not merely an excessive action of the motor centres on one side, but of the special centres for rotation. The two are, probably, closely related. The evidence of this is that in purely unilateral fits there is not necessarily, or even usually, any actual rotation, and in the cases in which this does occur the attacks are commonly severe and bilateral. But although actual rotation does not necessarily occur in unilateral fits (and so is not a simple consequence of unilateral discharge), it may occur at the commencement of fits which begin unilaterally, just as at the commencement of bilateral fits. In a patient, one of whose attacks I witnessed, the first event was that her head and eyes were suddenly turned to the right, then she turned round one and a half times, and would apparently have gone on turning had she not fallen; as she fell the spasm began in the right side, and afterwards involved the left.

The facial muscles pass into tonic contraction, greater on the side towards which the head deviates. It is usually most marked in the zygomatici, and hence the mouth is usually twisted towards one side. The orbicularis palpebrarum is sometimes little affected, sometimes considerably, and, in the latter case, more on the side towards which the head turns than on the other.

The posture assumed by the limbs under the influence of the tonic spasm varies in different cases. The most common posture of the arms is slight abduction at the shoulder-joint, flexion of the elbow-joint to about a right angle, very strong flexion of the wrists, while the fingers are flexed at the metacarpo-phalangeal joints, and extended at the others, the thumb being adducted into the palm or pressed against the first finger. The degree of flexion thus increases towards the periphery, but the fingers are usually in the position, not of simple flexion, but of interosseal flexion, *i.e.* that produced by action of the interossei—flexion at the metacarpo-phalangeal articulations, extension at the others, with the thumb pressed against the first finger or beneath it. The position, so far as the digits are concerned, is nearly that of grasping a pen, a posture of delicate co-ordination, established by constant repetition. The same posture is seen in many cases of athetosis and of paralysis agitans—a significant fact, indicating that the same nervous processes in the cortex, which are in over-action or unbalanced action in those diseases, determine the form of the spasm in many cases of convulsion. In some cases this posture is very constant, and the clonic spasm causes strong flexion of the metacarpophalangeal joints. Sometimes, however, the fingers are flexed at all joints, and the hand is "clenched." Excessive flexion of the elbows may bring the hands in front of the chest.

The legs are commonly extended, the feet inverted. The extension may not be complete; there may be slight flexion at the hip and knee-joints. Sometimes initial extension of the legs gives place to strong flexion in the later stages of the spasm. As a rule, however, the amount and extent of flexion is greater in the arms than in the legs; rarely it is the reverse. The spasm may be strictly symmetrical on the two sides, but more commonly there are slight differences in the degree of flexion of the arms, or of extension of the legs.

It may be noted that in this spasm we have reproduced the form of muscular action which is, physiologically, the most frequent and the most important. The chief use of the arms is in flexion, of the legs in extension. The nervous arrangements for these movements, respectively, will thus be the more developed. Moreover, the movements of the arms increase in complexity, and so in the development of their nervous arrangements, towards the extremity. Hence the flexor spasm increases towards the extremity. Further, all the more important and delicate actions (as writing, &c.) in which the fingers are concerned are those in which the interossei take the leading part. Strong flexion of the second and third phalanges is chiefly employed in comparatively coarse movements,—in providing, for example, an instrument by which the larger muscles of the shoulder and upper arm, &c., may do energetic work, as in lifting a weight. Hence we find that in the most common forms of convulsion the interosseal position is assumed in the spasm. Fits sometimes consist only of tonic extensor spasm, usually moderate in degree but much longer in duration than the typical form. Indeed, most attacks in which spasm continues for ten or fifteen minutes are tonic only. There is every gradation in degree from these to minor attacks with only transient slight rigidity.

Occasionally, instead of this combination of flexor and extensor spasm, flexion predominates throughout. The head is bent forwards, the arms are strongly flexed at the elbow-joints, and the legs at both hip and knee-joints, so that the knees are drawn up to the abdomen. In some of these cases also the fingers are strongly flexed at all joints by the long flexors. In some cases, indeed, the spasm in the arms is such strong flexion that the clenched fist is brought up against the neck, and even against the shoulders, while the legs may be so strongly flexed that the knees are near the face. Rarely, the legs may be in extensor spasm. The flexion of the limbs may give place to extension before clonic spasm comes on. The flexor attacks sometimes consist only of tonic spasm, but often change to clonic in the course of the attack. The head may deviate to one side, and occasionally there is alternate deviation. The diagnosis of this form from flexor fits of pure hysteria may be difficult, especially in boys, the more so if a true epileptic has minor seizures with violent post-epileptic hysteroid convulsion. These flexor fits often begin by falling forwards.

Another fact regarding the relation of flexion and extensor spasm is that it sometimes seems to depend on degree. In one patient the legs were always extended in slight attacks, but strongly flexed in severe ones. In connection with this we may note that extension of the legs is the chief effect of the influence of the spinal cord (as in standing), and it is intelligible that a more intense cerebral discharge should overcome the tendency of the spinal centres and produce flexion, which is the form of strong voluntary effort in many actions.

It may be noted that patients, before losing consciousness, sometimes have a sensation of flexor convulsion, although none exists. One patient, for instance, always woke up from his sleep with the sensation of being "twisted up into a ball with his legs round his neck," and then lost consciousness in a severe epileptic fit. All the time his limbs were extended and rigid. Another patient also woke up suddenly in the night, and immediately had a sensation as if his arms and legs "were all twisted up together," and then unconsciousness. His limbs were always straight out.

In rare cases the arms are raised up above the head at the onset of an attack; they may, indeed, be kept in that position through a distinct epileptic fit. Or, after being raised, they may be put straight forwards. In some patients, in whom the arms are held up throughout the attack, one leg, or both, is strongly flexed. In other cases of this kind the legs are extended and rigid. The raised arms may be flexed at the elbow-joint. In these attacks the head is often bent back.

In other fits, again, there is no flexion of the elbow-joints; the arms are straight out and rigid. In these the fingers are often strongly flexed at all the joints, but are rarely, if ever, in interosseal flexion.

The bilateral muscles of the body are involved in the tonic spasm as well as the limbs. The jaw is fixed and may deviate to one side; it is seldom widely lowered; the muscles of the thorax and abdomen also become rigid.

The tonic spasm arrests the respiratory movements and causes cyanosis and engorgement of the face. When severe, the aspect of the patient may be very alarming, and I have known even an experienced attendant to become so anxious for life as to commence artificial respiration,—so absolute and prolonged was the apnœa. In this case, three gasping inspirations renewed the supply of air, but deep stertorous breathing, with insensitiveness of the conjunctiva, continued for a quarter of an hour. It is probable, indeed, that it is by this mechanism that death sometimes occurs in a fit. The depression of the respiratory centre is so great that it cannot resume activity. Hence artificial respiration may be wisely adopted in such cases, when the tonic spasm has relaxed.

The violence of the tonic spasm is often very great; the shoulder may be dislocated by it, and in patients in whom this accident has once happened it is apt to occur again in subsequent fits. One patient sometimes dislocated the right shoulder, and sometimes both, and as is the rule, the dislocation was always subcoracoid. In this case the initial spasm was in the arms, which were first raised, and in slight attacks it was confined to them. The muscular spasm at the shoulders was probably unusually intense. The dislocation is on the side of the more severe convulsion, towards which the head deviates. This may however vary; one patient put out the left shoulder in the first fit, which occurred in the night. After two years he frequently dislocated the right shoulder. In a case recorded by Trousseau the occurrence of fits was not known until it was found that the patient's shoulder had been dislocated during sleep. This accident is not so surprising as it may appear, since the muscular action is abnormal and the usual antagonistic support may be absent. When it has once occurred the weakening of the ligaments must facilitate recurrence.

Dislocation of the lower jaw is a rare accident, and I have only met with a few instances of it. In a woman, nocturnal epileptic attacks began at twenty, and the mouth opened widely, with dislocation of the jaw, in the first fit, and the same thing happened in most subsequent attacks. In those in which it did not occur the tongue was bitten. There is preponderant spasm in the depressors in these cases. Moreover, the fact shows that the dislocation is the result of the tonic spasm, since the tongue is bitten in the clonic stage.

Clonic Spasm.—After the tonic spasm has lasted for a few seconds, or for one, two, or three minutes, the stage of clonic spasm comes on. Its onset is not sudden but gradual. In typical cases the tonic spasm becomes distinctly vibratory in character; the vibrations become less frequent and more distinct, until they consist of a series of clonic contractions. The remissions, as they become longer, become greater in degree, until at last they become complete intermissions, and towards the end of the fit the limbs are completely relaxed between the successive shock-like jerks. As the remission becomes greater the contractions apparently become stronger, but the increase in strength is apparent only, and due to the fact that each contraction starts from a greater degree of relaxation. The spasm ends by the remissions becoming longer and longer until, when they amount to one or two seconds, the jerks cease; but the last contraction is often as strong as any that have preceded it.

The muscles involved in the clonic spasm are usually the same as those which were most involved in the tonic spasm. As the clonic stage develops, the limbs retain the same position in which they were fixed by the tonic spasm, commonly with the arms in flexion and the legs in extension. During the intermissions the limbs may fall from the position they occupied before, but each clonic jerk tends to bring them back to it. In the most frequent posture, for instance, in each jerk the elbows are flexed, and the wrists flexed still more strongly, and the fingers jerked at the metacarpo-phalangeal joints.

The clonic spasm involves the muscles of the face, and also those of mastication, of the tongue, soft palate, and larynx. I have twice watched the soft palate jerking up and down synchronously with the other [illegible] during this stage that the tongue is bitten; the spasm in the muscles of the tongue pushes it between the teeth while the jaws are being jerked by the spasm in the masseter and temporal muscles. It is bitten on the side on which the spasm preponderates, and towards which the head deviates. Apparently the movement of the tongue is "contra-lateral," like that of the head, and it tends towards the side most convulsed.* The event occurs, at some time, in nearly half the cases with convulsion. The tongue may, however, be sometimes bitten during the stage of tonic spasm, being caught between the teeth as the rigidity comes on. It may thus be bitten in fits in which there is only tonic spasm. That tongue-biting is related to the spasmodic movement of the tongue bringing it between the teeth, rather than simply to the spasm of the jaw muscles, is illustrated by a case in which the teeth had been several times broken by the violence of the closure of the jaws, and yet the tongue had never been bitten. Of course the spasmodic movement of the jaws is the effective agent. In the case in which preponderant spasm of the depressors usually dislocated the jaw, the tongue was only bitten once, and then the jaws were not "put out."

Very rarely the cheek is bitten, occasionally together with the tongue and on the same side. A patient may, however, bite the cheek only in every attack. It is most rare for the lips to be bitten, though this is not unfrequent in hysteroid fits. These accidents are no doubt due to a peculiar combination of spasm in the facial muscles and in those of the jaw.

The clonic spasm affecting the muscles of the thorax causes the air to be forced intermittently through the lips, and frothy, often blood-stained saliva is then ejected, constituting what is termed the "foaming" at the mouth. The blood often comes from the bitten tongue, but sometimes seems to proceed from the mucous membrane during the vascular turgescence.

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* That is, it is protruded towards the side most convulsed, by the muscle of opposite side, just as, in paralysis, it is turned towards the paralysed side by site stronger muscles. So with the deviation of the head, spasm and being contrasted in their effects on these movements.

A still stronger proof of the violence of the spasm is afforded by the fact that a tooth is occasionally broken during the fit. This probably depends on some peculiar relation of the teeth, and perhaps on some slight deviation of the jaw by the spasm. Breaking of a tooth may occur when there is never tongue-biting—further evidence that the latter depends on the relation of the movement of the tongue and the jaw, and not on the degree of spasm.

Another form of clonic spasm is sometimes seen in epileptic fits. Instead of the shock-like clonic spasm, into which the tonic spasm gradually passes, the spasm consists of finer movements, of greater rapidity and less range, and is superadded to, instead of superseding, the tonic spasm. The latter continues, and the strained, rigid limbs present this fine rapid movement, almost like a coarse tremor, which maintains the same rapidity instead of becoming less frequent. It may even involve the jaw.* This form of spasm is often seen in the cases which present flexion of the fingers at all joints and tonic extension of the limbs. Sometimes it occurs in cases with flexor tonic spasm. Very rarely the two forms of clonic spasm are combined in different limbs.

In typical fits, as already stated, the tonic passes gradually into the clonic spasm. Very rarely the two are separated, and one well-marked case suggests that the tonic spasm may be reflex in nature. In this patient, whose minor attacks were characterised by a scream from epigastric pain, the severe attacks began with a similar scream, then she rose from the chair with arms stretched out, looked up and to the right, and turned round to the right. Then she fell limp, but in a few seconds general brief clonic spasm came on, in which she bit her tongue. It is conceivable that the intense sensory discharge may have inhibited the tonic stage, but not have prevented its sequel. Such cases are rare and should be carefully observed.

Although most attacks of epilepsy consist of both tonic and clonic spasm, in some fits there is but one form of spasm. As a rule, those which consist only of tonic spasm are general fits of slight severity. A patient falls unconscious, is rigid for a few moments, and then is better. Such cases are common as what may be termed the medium degree of attack, distinguished by the occurrence of such slight tonic spasm. In a girl of eighteen, with epileptic heredity, as the attack came on the arms were widely separated and stiff, the elbows slightly flexed, and the head was bent forwards. After a few seconds the spasm gradually relaxed, a vacant smile appeared on the face, and after a little mumbling and fumbling with the hands, she regained normal consciousness. In another case, a child, twenty to thirty fits occurred daily, consisting only of general rigidity, with the fingers flexed and the head turned to the right. About once a week a more severe fit occurred in which clonic convulsion followed the tonic spasm.

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* See Féré, 'Revue de Méd.,' Jan., 1891.

Occasionally more severe attacks consist only of tonic spasm, as the case of a boy of whose fits the following is a description:—His head was first turned to the right; then his arms became extended and rigid, the right being more abducted from the body than the left. Both elbow-joints were then flexed, and his fingers were flexed in the interosseal position. In a few moments the spasm ceased, lasting a little longer in the hands than elsewhere. There was no clonic spasm. He had also more severe attacks, consisting only of tonic spasm, but in them the arms were raised above the head; there was foaming at the mouth, and the tongue was bitten. These severe tonic fits (the "tetanoid epilepsy" of Pritchard) are not common.

In some patients who presented both forms of spasm, the tonic stage is very long, and the clonic stage is very short. For instance, a boy, in one attack which was witnessed, put his head a little back, his arms were put forward, and he became rigid. The trunk was twisted a little to the right, and the face was drawn towards the right side. This continued for about thirty seconds; there was then slight general clonic spasm for two or three seconds, and the attack was over.

Cases of severe convulsion, general from the first, in which the spasm is entirely or almost entirely clonic, are not very common, but are occasionally met with. Some of them are related to other forms of convulsion, especially to hysteroid fits. I once witnessed a striking example of this form of convulsion in a girl aged eighteen, who had also aortic regurgitation, with much hypertrophy of the left ventricle. She was distinctly hysterical and suffered from other pure hysteroid attacks, from frequent clavus and globus hystericus, and also from an attack of rhythmical hysterical chorea, which came on when another patient was admitted to the ward suffering from the same affection. But the attack I am about to describe, although differing from ordinary epileptic fits in several features, yet resembled them, in many important characters, most closely. She was sitting in a chair by me, answering questions, when suddenly, without the slightest warning, without any pallor of the face (which always had much colour), she fell headlong to the ground, striking her head against the floor with violence, and carrying the chair over with her in her fall. No mode of fall could have been more typical of an epileptic, as opposed to a hysteroid attack. Instantly all the muscles of her limbs and face were jerked in most violent clonic spasm, shock-like in character, exactly such as constitutes the second stage of an ordinary epileptic fit. It involved also the spine muscles, jerking the body backwards and causing opisthotonos. She was absolutely unconscious. Her face became livid and swollen, froth came from the mouth, and she appeared as if on the point of death, just as patients often do in severe epileptic fits. After a few minutes the clonic spasm ceased, and the limbs then became rigid, and the eyeballs converged just as in a hysteroid fit. The rigidity lasted but a short time, and then she lay motionless, and it was some time before any evidence of consciousness could be obtained. The characters of this attack will be again referred to.

In the slighter forms of bilateral fits, variations are sometimes presented in the distribution of the spasm. It may be limited to the arms and face, and the legs may be flexed though the arms are extended. The legs may be equally involved in flexor spasm, although the head and eyes deviate to one side.

The attacks which most frequently consist only of clonic spasm are slight fits of partial distribution, beginning in, and often confined to, one limb. The attacks of the most perfect type are those which are sometimes produced by organic brain disease, and Hughlings Jackson has proposed to designate them "epileptiform convulsions" as distinguished from ordinary epileptic fits. They are better designated "organic epilepsy" (see later). But such local attacks of clonic spasm sometimes occur in cases in which there is no other reason to believe that there is such disease as tumour, &c., and in which the variable commencement of the local fit, and other symptoms, preclude the supposition that the symptoms are due to any organic change. These local clonic convulsions are especially common in the upper limb, beginning in the hand. First the fingers begin to twitch, then the elbow is jerked, and then the shoulder. The attack may then cease, or the face or leg may be jerked in the same manner. Consciousness is often not lost if the spasm is confined to a single limb. Less commonly these attacks begin in the face, and still less commonly in the leg. Their march is described at a subsequent page.

It is rare for severe idiopathic fits to consist only of clonic spasm. A girl of twenty-four had such attacks. With a scream she rushed out of bed, falling unconscious on the floor, with eyes turned upwards, and arms and legs jerking in clonic spasm, with imperfect relaxation between. After one minute the clonic spasm ceased and the limbs relaxed, but the eyes were directed to the right for three minutes.

When a severe attack begins by local clonic spasm, this, when it reaches a certain extent and degree, may pass into tonic spasm, followed, at a later stage, by secondary clonic spasm. In such cases the period at which the tonic spasm comes on, and its duration, vary: if the attack is severe, it may come on early; if slighter, there may be only a little tonic spasm in the middle of the fit. An instance of the latter form was afforded by a girl aged ten, who, while I was looking at her, suddenly turned her head to the right, then turned round to the right and fell. As she fell, the right arm began to twitch at the shoulder and elbow-joint; the clonic spasm did not involve the hand, the fingers of which were extended and still; presently the face began to twitch on both sides, and both legs were similarly involved, with the left arm. Then all parts were fixed in tonic spasm, which lasted for a few seconds, and was again succeeded by clonic spasm.

In fits which begin locally, the spasm is not always clonic in the commencement; it may be at first tonic and afterwards clonic, as in a general attack, and this although the fits are very limited in range. In one patient, for instance, the attacks were limited to the right arm and right side of the face, and commenced by tonic flexion of the fingers, first at the metacarpo-phalangeal joints, and then at the other joints, the flexion being greatest towards the ulnar side. The thumb was inverted or pressed against the first finger. Next the elbow-joint was gradually flexed, and the arm adducted by the pectoralis, so that the hand was brought in front of the chest. After the arm had got into this position, the tonic gradually passed into clonic spasm exactly as in an ordinary epileptic fit. This patient had also some attacks which began in the face, and a few which began in the right leg. There was a probability (although not conclusive evidence) that he was the subject of cerebral tumour, but his seizures are here mentioned because they are particularly instructive as an illustration of the characters of this form of seizure. Different parts, as the face and arm, being affected in succession, often presented, at the same moment, different stages of convulsion. For instance, when the face was affected first, and the arm secondarily, there was frequently clonic spasm (second stage) in the face, and tonic spasm (first stage) in the arm. In slight attacks involving face and arm, the leg often escaped altogether, but in severe attacks it was involved in the tonic and clonic spasm. In less severe seizures the leg was the seat of the tonic spasm only, although the face and arm presented both forms of spasm. In some of these attacks a change in the form of tonic spasm in the leg was observed to occur at the time of the change from tonic to clonic spasm in the arms. The leg was always rigidly extended, and at first the foot was inverted and the great toe pointed downwards and inwards. When, however, the change in the form of the spasm occurred in the arm, the foot ceased to be extended and inverted, the ankle-joint became flexed and the great toe over-extended so as to point upwards.

It is instructive to compare with this change in the form of tonic spasm, from extension to flexion of the ankle, synchronous with the change from tonic to clonic spasm in the arm, the characters of the tonic fit mentioned on the last page. In this during the course of the fit there was a corresponding change from extensor to flexor spasm.

March of the Convulsion.—Considerable interest attaches to the march of fits which begin locally, and of which the case just mentioned is an example. It is scarcely possible to consider this subject without referring to certain pathological questions which are involved, and which would find a place more logical, although less convenient, in the chapter on Pathology. The theory here assumed, that the discharge in epilepsy originates in the cerebral hemispheres, will there be further considered.

Hughlings Jackson has drawn attention to the frequency with which the fits begin in the hand, and has associated with it the law that the spasm most frequently begins in small muscles which are put to the most varied uses, and he has brought forward also the hypothesis that these small muscles are represented by small cells, and that such cells are less "stable" than larger ones. But it is important to note that (as stated in the description of modes of onset) a large number of fits beginning in the hand commence, not by a motion, but by a sensation. No doubt the sensory centres in which the hand is represented are much more highly developed than those in which the upper part of the arm is represented. But the law holds good only generally, not universally, with regard to either motor or sensory auræ. The thumb and forefinger are the parts put to the most varied uses, but initial spasm, though it sometimes occurs in them, occurs more frequently in some other part of the hand, and an initial sensation is much less frequently felt in the parts of the hand in which sensation is most highly developed—the palmar aspect of the fingers—than in some other part, as the palm itself or the wrist.

Moreover, cases are not rare in which the spasm begins in the larger muscles of the shoulder or hip. I have mentioned a case in which the convulsion began in the shoulder. In another patient, whose attacks were carefully watched, twitching began at the left shoulder, and the spasm descended the arm; then it began on the left side of the face, and finally affected the leg. In the leg it is very frequent for the first spasm to begin in the large muscles of the hip or thigh. But it is to be remembered that elaborateness of movement is much less developed in the foot than in the hand.

The march of the spasm in the cases in which the commencement is deliberate and local, has been already considered at some length in the account of the modes of the onset. It usually extends rapidly through one side, and then affects the other. It is partial, confined to one side, or to part of one side, less frequently in idiopathic epilepsy than in the convulsions which result from organic brain disease. In many cases it has begun to lessen on the side first affected before the second side is involved, so that it may be in a different stage, at the same time, on the two sides. When the side first affected is in clonic spasm, the side affected secondarily may be in tonic spasm. The extension to the second side is marked, as already stated, by a turning of the head and eyes away from the side first affected. The cessation of the fit is usually in the order of invasion. It ceases first in the part first affected.

A good example of this deliberate extension to the other side was presented by a girl of thirteen, who had suffered from fits since four years of age, and came under observation during a period of status epilepticus. In the slighter fits there was first violent twitching of the left side of the face, followed by deviation of the head and eyes to the left, changing to the right after thirty seconds. In more severe attacks, the spasm in the face was followed by strong flexion of the elbow and abduction of the arm, succeeded by adduction as the tonic spasm became clonic. When this was ceasing at the elbow and wrist, the leg became extended and then jerked. The right side of the face then began to twitch, and the convulsion passed through the right side in the same way as it had traversed the left.

The relation of the spasm to the muscles of unilateral and of bilateral use is a point to which attention has been called by Hughlings Jackson. In a unilateral convulsion, for instance, the respiratory muscles on both sides of the chest may be equally affected. A unilateral fit beginning in muscles of perfect bilateral use commonly commences in both. I have recorded[*] a case of convulsion from cerebral tumour (situated in the white substance of the hemisphere above the lateral ventricle), in which the convulsion, although unilateral, began in both frontal muscles. The orbicularis oris is a medial muscle of bilateral use, and a unilateral convulsion commencing in this muscle begins in the whole.[†]

The theory of Broadbent,[‡] to explain the escape of the bilateral muscles (of thorax, abdomen, jaws, eyelids, eyes, &c.) in hemiplegia, affords an equal explanation, as Hughlings Jackson has shown,[§] of their involvement in spasm. The theory is that the commissural connection between the nuclei for these bilateral muscles is so close that the two nuclei are practically fused into one, and can be called into action from either hemisphere of the brain. Underlying the theory of Broadbent is the wider and more important hypothesis that the bilateral muscles are represented in each hemisphere of the brain much more equally than the muscles of chiefly unilateral use. We do not yet know in what manner this is subserved, and it may be by a direct connection of these muscles with the hemisphere on the same side, by fibres which do not decussate, as well as by a connection between the two hemispheres. The essential element is the bilateral representation of the corresponding muscles that habitually act together, so that, in proportion to their association, they can be innervated from either hemisphere.

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[*] 'Brit. Med. Journal,' September 26th, 1874.

[†] Hughlings Jackson, 'Med. Times and Gazette,' January 6th, 1872. I have also met with a similar case.

[‡] 'British and Foreign Med.-Chir. Review,' April, 1866.

[§] 'Med. Times and Gazette,' August 5th, 1868; and "Localisation of Movements in the Brain," 'Lancet,' February, 1873.

A discharge of the motor centres in one hemisphere causes a convulsion on the opposite side. If this is slight, only the muscles of unilateral use may be involved. In such convulsion, the muscles of bilateral use (as the muscles of ordinary respiration) may escape, probably because the very equality of respiration in the two hemispheres of the brain involves a less degree of representation in either. If the discharge is greater in degree, it involves, besides the unilateral muscles of the opposite side, the bilateral muscles of both sides, in proportion to the degree of functional association. The muscles of the thorax are affected equally. The leg on the side of discharge may be affected, but in a slighter degree than the other. The arm on that side usually escapes. When the spasm spreads to the whole of the second side, it is doubtful whether the discharge is limited to the one hemisphere.* The character of the convulsion, in cases in which the spasm has involved first one side and then the other, seems to suggest strongly that there is an extension of the discharge from one hemisphere to the other. In the early stages of the fit there is a deviation of the head and eyes to the side first affected, and as the spasm is lessening on this side there is a similar deviation of the head and eyes to the side secondarily affected, and the convulsion on this side passes through the same stages as on the other, on which it has often ceased when it is at its height on the second side. The corpus callosum certainly connects corresponding convolutions of the two hemispheres; it subserves associated action, and it has been demonstrated by experiment that through its fibres the motor centres of the cortex may be called into action.

It may be urged that in idiopathic epilepsy the affection of the two sides is synchronous, and that therefore there cannot be a passage of the discharge from one hemisphere to the other. But the instantaneous involvement of all the muscles of one side may occur from spread of local discharge. In some cases minor attacks begin locally and spread slowly, while severer attacks seem to begin simultaneously in all the muscles. In the latter case we are justified in assuming that there is a spread of discharge through the centres, which occurs too rapidly to be observed. There is no greater difficulty in admitting the instantaneous spread of an intense discharge to the opposite hemisphere than in admitting its instantaneous extension through the centres of one hemisphere. So rapid is the transmission of nervous impulses, that centres structurally connected by fibres which extend from one hemisphere to the other may be in as intimate functional connection as centres which lie in juxtaposition. We have abundant evidence of this in the processes concerned in vision.

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* See, however, the experiments of Luciani ('Rivista Sper. de Frenatria,' vol. iv, 1878, p. 617), in which he succeeded in producing general convulsions by stimulating the leg centre in one hemisphere, all other cortical centres having been removed on both sides, which show that general convulsion may be produced by a unilateral discharge, possibly, as he supposes, by the subordinate agency of the medulla oblongata. But the transfer of the inference to man may not be justifiable.

That this theory of the process of the epileptic fit is correct, is, I think, proved by a very important case which has been published by Oebeke,* and which also, as its narrator points out, is of not less value from the evidence which it affords that the convolutions are the seat of the discharge in idiopathic epilepsy. A patient who had been liable to general epileptic fits from birth, was seized, in adult life, with left hemiplegia, due, as was afterwards discovered, to a hæmorrhage in the central ganglia of the right hemisphere. The epileptic fits continued to occur after the onset of the hemiplegia, but affected only the unparalysed side. The arrest of conduction from the right cortex prevented the effects of its discharge, showing that the convolutions of one hemisphere cannot act on the limbs of the opposite side, at least to a considerable degree, through inferior commissural connections.

The more rapid the development of the spasm in a fit beginning unilaterally, the quicker and more equal is the involvement of the side afterwards affected. As an exception to the rule that severe unilateral spasm commonly spreads to the other side, and affects it considerably, a very unusual case deserves mention in which, in a very long continued attack, one side was involved almost exclusively, the other being the seat of slight convulsion from time to time. The duration of the attack (twenty minutes) was also very remarkable. The patient presented no evidence of organic brain disease.

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* 'Berlin. klin. Wochenschrift,' 1880, No. 37.

The patient was a boy ten years of age, who had been subject to fits since the age of six. There was no inherited tendency. The attacks were preceded by an aura which commenced at the lower part of the stomach with a "winding up," and the sensation seemed to ascend to the top of the head, when he lost consciousness. Several attacks which were seen were brief right-sided fits, in which the arm and leg became rigid, the latter being strongly flexed. He also had two longer attacks, one of which was watched by Dr. Beevor, then resident medical officer, who noted, about three minutes after the onset, that the patient was quite unconscious; the head and eyes were turned to the right, the face was jerked on the right side, the right arm was strongly flexed at the elbow, and jerked in violent flexor clonic spasm. The fist was clenched. The right leg was extended and rigid, and the seat of fine tremor. The respiratory muscles were convulsed synchronously with the arm. The pupils were both widely dilated. After this convulsion had continued on the right side for ten minutes (by the watch), and the face, at first red, had become cyanotic, the clonic spasm commenced in the left arm and leg, the latter being flexed at the hip and knee-joints. After a few minutes it ceased on the left side, continuing on the right. Nineteen minutes after the onset the convulsion ceased in the right side of the face, and at twenty minutes in the arm and leg also, and then the left arm and leg were again slightly convulsed for a few minutes. In twenty-five minutes there was again slight rigidity in the left arm and leg, with deviation of the head to the left. In two or three minutes more this also ceased, and there was no return.

Sensation of Convulsion.—Little is known of the sensation which attends severe convulsion, since the patient is almost always unconscious. Slight clonic or tonic spasm is often unattended by pain. When considerable it is usually very painful, and sometimes a slight amount of spasm is attended with considerable suffering. Thus one patient described the sensation before he lost consciousness "as if his arms were being torn off." Another, who had unilateral fits without loss of consciousness, said that "if he put his arm into a furnace it would not equal the pain." Another, a girl of twenty-five, who had had fits since the age of thirteen, was often waked in the night by the attack. She would find the head turned to the left, and the left arm "twisted," and she said that she "could not think that the pain of the arm being cut off would be more severe than that she then felt." A patient with chara [illegible] eptic fits first felt the head and eyes turning to the left, and then as if both arms were being twisted and wrenched off in the middle of the upper arm. It is probable that in some cases in which the pain is very severe, and the spasm moderate, the sensation is not due to the latter, but to the extension of the discharge to the sensory centres related to those for movement.

Pupils.—At the onset of a fit the pupils have occasionally been found contracted. Dilatation, however, quickly occurs, and is present as soon as the tonic stage is well established. It is rare for the pupils to be seen in any other condition than dilatation. This continues till the fit is over. While dilated they seldom act to light. When the patient begins to exhibit slight signs of consciousness on an attempt to rouse him, the dilatation ceases.

Loss of action to light is not invariable in fits of moderate intensity, although opportunities for precise observation are rare. In a girl of fifteen, an attack of tonic spasm—the fists were clenched, arms drawn up, head and eyes turned to the left, and a few jerks at the end, with complete loss of consciousness; the pupils were dilated, but acted to light on being carefully tested in the middle of an attack.

At the end of the fit, usually a few minutes after the cessation of the convulsion, the pupils sometimes exhibit a remarkable oscillation. It was first pointed out by Reynolds,* and attention has been specially directed to it by Clouston and Echeverria. It is not a constant phenomenon, being more frequently absent than present. There are alternate contractions and dilatations, repeated every one or two seconds, for a few minutes.

Reflex action is abolished in all severe fits, but its investigation is a matter of difficulty on account of the spasm. The absence of reflex action of the iris has been just spoken of. The conjunctiva may be touched without any reflex contraction of the orbicularis. The loss continues until a few minutes after the convulsion is over, but its condition, and that of the deeper reflexes, after the attack, will be described presently.

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* 'Epilepsy,' &c., 1861, p. 112.

Sphincters.—During a fit there may be involuntary passage of urine and fæces, the former much more frequently than the latter. It is not merely the result of unconsciousness, since it occurs usually in some patients, never in others, although their attacks are equally severe. Further, urine may be passed without the patient being at the time actually unconscious, as in a case which will be mentioned in the description of minor fits; also, the urine may be ejected with great energy, even in attacks in which slight tonic spasm was insufficient to cause a fall. The event seems thus to be part of, or related to, the convulsive action of the fit. The same conclusion is suggested by the occasional relation of the act of micturition to the aura of a fit, as pointed out on p. 61. Passage of urine or fæces is rather more common in fits which come on during sleep than during the waking state. It is generally a symptom of a severe fit, being accompanied by tongue-biting in the majority of the cases in which it was noted (60 per cent.), while micturition without tongue-biting occurred in only 25 per cent. of the cases in which the presence or absence of the symptom was recorded. It may be invariable in nocturnal fits, although not during fits in the day. Yet it may sometimes attend diurnal fits, and not those in the night. It is also common for pale, limpid urine to be passed immediately after a slight attack. Emission of semen is said sometimes to occur towards the end of the tonic stage, but is very rare; indeed, its occurrence is not certain.

Vascular System.—The pulse may be feeble at the onset, but I have never noted an actual initial failure. It is generally unaffected at the commencement. A tracing published by Voisin* shows that the heart's action may be perfectly normal during the stage of the aura. A case, observed by the late Dr. Moxon, with initial failure of the pulse, which led him to genralise too widely, was certainly exceptional. One instance has been described to me by Dr. Money, in which the absence of failure of the heart was well observed. It occurred in the course of an examination for life assurance.

"I was examining the heart, which was beating vigorously, when I noticed some unsteadiness of his body. His face had become pale, with a vacant look. He fell forwards and was laid down, somewhat stiff; the face became somewhat dusky. After a few seconds the arms and legs were jerked, for perhaps six times, with the right side of the face. The pulse was full and slow at the wrist when he was first laid down, and continued so, getting slower but keeping full, until he opened his eyes after two minutes of unconsciousness. He was at first a little dazed, but soon regained a normal state."

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* 'Dict. nouv. de Méd. et Chir.,' art. "Epilepsie," tom. xiii. p. 584.

As the muscular spasm becomes considerable, the pulse is increased in frequency and often also in force. It may be 120—140 per minute. The frequency gradually lessens after the termination of the fit, and it then usually becomes feeble and small.

The colour of the face at the onset varies in different cases. It may be pale, but the pallor is far less frequent than is currently asserted. It is sometimes quite unchanged, and in some cases the face is flushed at the onset. As Voisin correctly states, the pallor may be considerable after the tonic spasm is established, although there was none at the onset. After the tonic spasm has lasted a short time, ten or twenty seconds, the face becomes congested, and then cyanotic. The venous congestion may be extreme, and the bloated dusky tint of the features, distorted by spasm, renders the aspect of the patient most alarming to those unaccustomed to the disease. With the increasing remission of the spasm, air becomes changed in the lung, and the cyanosis lessens. During the course of the convulsion the skin often becomes covered with sweat.

Ophthalmoscopic examination during a fit of idiopathic epilepsy has rarely been satisfactorily effected. My own observations relate chiefly to cases of convulsions beginning locally, a class of fits in which there is rarely any initial pallor of the face. In several of these cases I have watched an artery by the direct method from before the commencement to the end of the fit (in one case during a very severe unilateral convulsion) without detecting the slightest diminution in the size of the vessels. In one case of idiopathic epilepsy a fit came on during ophthalmoscopic observation, and no change in the vessels was visible at the onset. During the stage of cyanosis, the veins of the retina always become distended and dark. During the status epilepticus, in which fits occur in rapid succession for several days, I once observed congestion of the discs with slight œdema, which passed away after the cessation of the series of fits. During the intervals of fits the ophthalmoscope does not, according to my experience, reveal any abnormal appearances which can be associated with epilepsy. The elaborate descriptions that have been given of slight changes are due to ignorance of the varieties of appearance presented by the optic discs in health.

EXCITANTS OF ATTACKS.—In some patients attacks may be excited by special influences. Emotion has much less effect in bringing on epileptic than hysteroid seizures, but it is sometimes distinct in the former. Sudden noises occasionally produce attacks, usually of minor character. In one case, for instance, in which the severe attacks were certainly epileptic, and were attended with tongue-biting, minor attacks, consisting of a sudden swoon, were readily produced by a loud noise. A still more striking case was that of a young man whose attacks began by a sensory aura in the hand, passing up the arm, and in whom minor attacks, consisting of this sensation only, could be at any time produced by a sudden loud noise, such as slamming a door. A sudden "startling" noise, as the adjective denotes, may cause, normally, a momentary discharge of the motor centres, and it is therefore not surprising that it should excite a pathological discharge when there exists a morbid instability of tissue. In one patient, with a marked susceptibility to noises, attacks were also induced by any unpleasant smell, especially that from a drain—an effect also intelligible when we consider the inhibitory effect of fainting, and the motor disturbance of vomiting, which are also induced by odours.

In very rare instances the influence of light seems to excite a fit. I have met with two examples of this. One was a girl of seventeen, whose first attack occurred on going into bright sunshine, for the first time, after an attack of typhoid fever. The immediate warning of an attack was giddiness and rotation to the left. At any time an attack could be induced by going out suddenly into the bright sunshine. If there was no sunshine, an attack did not occur. The other case was that of a man, the warning of whose fits was the appearance before the eyes of "bright blue lights like stars—always the same." The warning and a fit could be at any time brought on by looking at a bright light, or even at a bright fire. The relation is in this case intelligible, since the discharge apparently commenced in the visual centre. The same relation was presented by the patient already referred to, with an auditory aura, which could be always induced, with the sensation of a fit coming on, by a low organ note. Such a relation to the aura, however, is not invariable. In a girl, who never experienced an olfactory aura, attacks were often induced by a smell, sometimes pleasant, more often unpleasant.

In some, also rare, cases attacks can be brought on by voluntary motion. A curious instance of this was presented by a boy in whom, at one time, any passive movement of the trunk which involved a movement of the spine, at once brought on an attack of general tonic spasm, in which the legs were extended and the arms irregularly flexed just as in epilepsy, but without any clonic spasm. During the attack, which lasted only fifteen seconds, there was complete unconsciousness. Afterwards an attack occurred whenever, after remaining still, he attempted to walk, but the attacks were then much slighter, and consisted only in sudden irregular fixation of the limbs without loss of consciousness. In another patient who had also severe epileptic fits, minor attacks, consisting only of giddiness and staggering, were sometimes excited by sudden movements. A similar effect was observed in a girl of thirteen who for two years had had attacks of a very unusual character, brought on by movement after sitting still. On rising from a chair, for instance, after taking four steps she suddenly made a quick movement forwards; the right arm was raised to the head, and the other put down and back; the fingers of each hand were strongly extended, and the right hand made scratching movements, and did actually in some attacks scratch the face severely; her face assumed a terrified expression, the eyes wide open, the tongue protruded to the left and grasped by the teeth, but not bitten; the face was flushed at first, and gradually became pale; the pupils were widely dilated, much more so than was habitual. I was once able, during an attack, to test the action of the pupil by direct sunshine; it was quite motionless. The attack lasted about a minute; she was confused for a moment or two; then she was quite well. But after some attacks she swung her right arm round as if turning a mangle. At a later period this co-ordinated movement after the attack did not occur, and the fits came on independently of movement. They were certainly epileptic in essential nature.

Such induction by voluntary movement is less rare in cases of organic disease causing convulsions, especially in such diseases as cerebral tumour.

Very interesting is the effect of sudden muscular tension, or the vibration of tense muscles produced by percussing their tendons, in exciting attacks. The effect is chiefly seen in such cases of organic disease, with convulsions beginning locally, and especially when they commence in the leg. In these cases the tap on the patellar tendon, or the sudden flexion of the ankle in an attempt to obtain the foot-clonus, may set up an attack commencing in the leg. In some cases of this character, indeed, even gentle passive movement of a weakened limb may excite the commencement of an attack. In one instance of this the right arm had gradually become weak and paralysed; there were frequent convulsive attacks, beginning in the right hand. The arm was rigid, and any attempt at passive movement, even the muscular tension of allowing the arm to hang down, brought on the sensation of a commencing fit. Voluntary movement of the fingers had a similar influence. Such movement had the same effect in a girl in whom these fits commenced after typhoid fever, and were probably due to a cortical lesion occurring in the course of that disease.* This effect is comprehensible when we consider that the disease of the motor centres has weakened the resistance that limits and conditions voluntary action. The activity induced by the will spreads in them in an abnormal manner, and to an abnormal degree. In the adaptation of muscles to posture there must be a cerebral as well as a spinal process, an effect on the motor centres of the brain as well as on those of the spinal cord, produced by the afferent impulses from the muscles, which enables us to understand that such impulses may excite discharge.

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* A similar effect of voluntary movement has been described by Marie ('Epilepsie,' &c.).

Phenomena analogous to the "epileptogenic zone" of Brown-Séquard's guinea-pigs—the production of attacks by irritating some part of the skin—are almost unknown in idiopathic epilepsy.* In a man aged thirty-three, who was subject to right-sided fits since a fall on the head at twenty-two, the attacks began deliberately by spasm in the right arm, and they could be at any time produced by touching the skin at the upper edge of the *left* scapula. In another case, in which the head was struck in a fall out of a carriage at twenty-two, stunning the patient for an hour, attacks began after a few days, consisting of brief tremulous flexion of the fingers of the right hand. The spasmodic attacks increased to flexion of the wrist, then the elbow was also bent, and afterwards the arm was carried back behind the trunk so that the hand was brought towards the back of the head, and this was bent slightly backwards. Loss of consciousness then occurred, and the convulsion spread to the left side. But an attack could always be induced by a tap on the edge of the right scapula. Such cases are at present beyond clear interpretation.

Arrest of Fits.—Sometimes attacks which begin deliberately can be cut short, and the means by which their arrest can be effected are of considerable pathological interest and some practical importance. Attacks which begin by a general or bilateral aura, or by the epigastric sensation, can rarely be arrested, but now and then they may be stopped by some muscular exertion, as by walking quickly about the room, which seems to relieve the tension in the motor centres, or by a strong sensory impression, such as ammonia to the nostrils, or taking a spoonful of common salt into the mouth, which seems to excite sensory centres of wide relations, and inhibit the commencing discharge. Some cases of deliberate onset may be arrested by the inhalation of nitrite of amyl. The effect of this is to flood the brain with arterial blood, and increase the pulsation of the small arteries in a sudden degree, forcibly influencing consciousness. We do not know by what mechanism this sudden alteration of normal conditions arrests the commencing discharge. The result is unquestionable in many cases, but unfortunately there is seldom time for its adoption in the forms of general convulsions to which it is chiefly suited.

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* 'Medical Ophthalmoscopy,' p. 256.

The attacks which can be most frequently arrested are those which begin by a unilateral peripheral aura, especially those commencing in the hand or the foot. The most common mode by which arrest is effected is by the application of a ligature around the limb, above the part convulsed. This method is due to Pelops, the master of Galen, who was led to employ it by his theory already mentioned, that the aura consisted in the ascent of some vapour, analogous to a venomous poison, up the vessels, and the ligature was applied to arrest the ascent of this, just as it would arrest the ascent of the poison of a snake-bite. It is a striking instance of the way in which a visionary pathology may, as it were, coincide with morbid facts of a different nature, and the treatment, deduced from a mistaken theory, may prove effective. The ligature was frequently employed by physicians of the sixteenth and seventeenth centuries with the same object—to arrest the progress of some influence along the vessels, or, when the theory changed, along the nerves. This explanation has, however, been untenable since the demonstration by Odier in 1811, that this method of arrest is equally efficacious in convulsions which are due to organic brain disease. This fact shows that the arrest must be effected, not in the limb convulsed, but in the centre in which the discharge is occurring, of which the local convulsion is the outward manifestation. The strong peripheral impression on the limb above the part convulsed seems to alter the resistance in the nerve structures of the related part of the brain, and thus arrests the spread of the discharge. The effect of the ligature must first be exerted on the sensory elements, and through this on the motor structures. The two are intimately connected, and their mutual interaction must be constant, sensation and movement in any part being closely related. Hence the condition of the motor centre is no doubt readily influenced by that of the sensory centre. The effect is doubtless produced by a process connected with that by which a sensory discharge causes temporary motor weakness. A fit may be arrested by the ligature whether it begins by a sensation or by a motion, *i. e.* whether the sensory or motor centre leads in the discharge. If the commencing fit, the sensation or the spasm, has got beyond the part to which the ligature is applied, the attack is not arrested. The reason why a ligature is especially effective is probably partly because it is readily adopted, and partly because the cutaneous stimulation is applied to the entire circumference of the limb, and so seems to influence the whole extent of grey matter in which the discharge is advancing. Occasionally, however, a more limited cutaneous stimulation, as a pinch or a prick, has a similar effect.

It is instructive to note the antagonism which seems to exist between those afferent impulses that are excited from the surface in cutaneous stimulation, and the afferent impressions which come from the muscles when they are extended, or the tension is suddenly increased, as in the attempt to obtain the contractions commonly called "tendon-reflexes," the knee-jerk and foot-clonus. It has been already pointed out that one effect of this deep stimulation may be to excite a fit. On the other hand, the effect of the stimulation of the skin may be to arrest the fit. Some years ago a man was under my care whose fits began in the foot. The sensation with which the fits commenced could be at any time produced by percussing the patellar tendon, and then could be arrested by pinching the skin of the leg. A similar antagonism may be sometimes seen in disease of the spinal cord, attended by a spasmodic state of the limbs—spastic paraplegia. The paroxysms of spasm, broken by tremor, which often occur in that disease, may usually be at once arrested by a strong cutaneous impression. Thus in both cord and brain the "discharge" may be arrested by the same means.

The repeated arrest of fits by the ligature may produce a permanent effect. A patient had fits that commenced in the hand by a sensation which passed up the arm, and could be arrested by a ligature applied just above the elbow. After employing the ligature in this manner for some months, he found that the fits, commencing in the same way, stopped of their own accord at the spot at which they had been repeatedly arrested by the ligature. They had never stopped thus before the patient commenced the use of the ligature, which appeared to have produced, in time, a permanent increase of resistance at the related part of the unstable nerve tissue. Afterwards he found that a slight "discharge" stopped at the place at which the ligature had been applied, while a strong one overpassed it and progressed to a fit, unless he had applied the cord. This was still effectual, and he could tell, by the degree of the sensory warning, whether it was likely to pass the arrest-place, and whether he need have recourse to the ligature.

If this explanation of the mode of action of the ligature be correct, it is evidently important that every fit should, if possible, be arrested, since a single passage of the barrier by the discharge might, so to speak, sweep away any functional resistance which had been established there, and render the subsequent arrest of fits more difficult. An illustration of this is afforded by the case of cerebral tumour recorded by Odier and before referred to.* The convulsive attacks were at first local, beginning in the little finger of one hand, and ascending at first only to the wrist, then to the elbow, and ultimately to the shoulder. The patient learned to arrest them by a ligature round the wrist, and did so for a long time. One day he omitted to apply the ligature, and the attack went on to a severe general convulsion. The commencing fits could never afterwards be arrested by the ligature.

The fits which begin by spasm may often be effectually stopped in another way—by forcibly preventing the movement, as was pointed out by Aretæus,* and especially insisted on by Maisonneuve in the beginning of this century. For instance, in one patient the attacks commenced by forcible closure of the right hand. If he could succeed in forcing the closed hand open by the other hand, the fit always ceased; if he failed the spasm went on up the arm, and he lost consciousness. In another patient the fits began with cramp in the thigh, which passed down to the heel, the leg quivering. As soon as it reached the heel, the leg flexed forcibly on the thigh, and the thigh on the body. If the patient could succeed in preventing this forcible flexion of the leg, by keeping it down, the fit ceased. If he failed to do this, the attack went on to a severe convulsion. In another case, in which the initial aura was a bright dazzling light, this was followed by a deviation of the head to the right. If he could bring it back by a voluntary effort, the attack ceased.

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* 'Man. de Méd. pratique,' 1811, p. 130.

To this influence also an analogue may be found in the action of the spinal cord. The muscular cramp, such as occurs in the calf in the frequent experience of most persons, seems to be due immediately to the over-action of motor nerve structures of the spinal cord. If the contraction of the muscle can be prevented, as by pressing the ball of the foot against some object when the cramp is commencing, it passes off. Here we have (as Herpin† pointed out) the same phenomenon. The extension of muscles arrests, no doubt by its influence on the centre, the discharge of the motor elements of the spinal cord, just as it does the discharge of those of the brain in a commencing epileptiform seizure.

But we have seen that, if there is no fit in progress, muscular extension, and the vibration of tense muscles, may set up an attack. Here we have an antagonism between the action of the same stimulus, probably in different degrees of intensity, acting in different states of the nerve-centre. This form of antagonism is familiar to physiologists. A slight peripheral impression, for instance, will set up a reflex action, while a strong impression of the same kind will arrest or prevent all reflex action. Passive muscular tension excites tonic contraction in a muscle (as Tchirjew has shown), and this action may in abnormal conditions be excessive, as in the "myotatic" contractions (so-called tendon-reflexes). It has an effect on an unstable cerebral centre similar to its effect on the spinal centre, and excites the epileptiform discharge. But if this centre is in process of discharge, as in cramp, a strong extension of the contracting muscle may arrest the discharge in the related centre. The afferent nerves, it is to be noted, commence in the interstitial fibrous tissue of the muscle, and seem to be stimulated especially by extension.

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* "Whenever the disease occurs, and has already seized the finger, or is commencing in any part, [the patients] having from experience a foreknowledge of what is to happen, call, from among those who are present, upon their customary assistants, and entreat them to pull aside and stretch the affected member."—Aretæus, Syd. Soc. trans., p. 244.

† 'De l'Épilepsie,' 1852, p. 603.

In some cases, rubbing the limb in which the attack is commencing will arrest the fit, a fact which patients often discover for themselves. Almost all forms of spasm, even the "late rigidity" of hemiplegia, and the rigidity of "spastic spinal paralysis," may be lessened by rubbing, and we have another illustration of the fact that influences which lessen the discharge of force from lower centres have a similar effect on higher centres.

ABORTIVE ATTACKS.—The phenomena which attend the sudden termination of threatened attacks are sometimes peculiar and instructive. In one case, a cold and "shaky" feeling in the legs passed up the back to the head, which felt as if it would burst; when this sensation had become intense, there was a sudden flow of saliva, and a watery discharge from the nostrils; then there was a copious flow of tears for a few seconds, and the sense of fulness in the head suddenly ceased, and also the secretion from the mouth and nose. In some cases the act of micturition seems to arrest an impending fit; so also does a copious eructation from the stomach when there is no reason to refer the attack to gastric disturbance.

CONDITIONS AFTER ATTACKS.—*Sleep and Headache.*—The coma into which the patient usually passes at the close of an epileptic fit often continues as heavy sleep, lasting for a quarter of an hour or longer, sometimes for hours. Its intensity is often profound. It constitutes one of the dangers to life that epilepsy entails. If the mouth is brought against the pillow, by the patient turning over to the prone posture, the additional asphyxia may end life without any opportunity being afforded for the return of consciousness. So also the added influence of even a quarter of a grain of morphia, given, for instance, before the attack, may so intensify the post-epileptic coma as to render it a lethal narcosis. The sleep is usually followed by a severe headache, general, lasting for several hours more, and often continuing for the rest of the day. Both sleep and headache may be absent. Occasionally the sleep is not succeeded by headache, but if the patient is roused, and is not allowed to sleep, the pain in the head is severe. Often, indeed, there is a distinct tendency for headache or sleep to replace each other; a patient who sleeps will not have headache, and one who does not sleep has pain in the head for hours. So also fits may be succeeded by transient mental disturbance when there is no sleep, but, after even half an hour's sleep, normal consciousness may be gradually but quickly regained. Sometimes a patient has a brief period of consciousness between the attack and the sleep. One patient described a sensation immediately after the fit, like a bell in the back of his head, which "seemed to send hiim off to sleep."

Paralysis.—A fit of epileptic type may leave transient paralysis, very rarely general, commonly of one half of the body, or of still smaller range, affecting a limb only. The form and nature of the paralysis vary essentially in different cases, and by many writers the varieties have not been distinguished. The first and most common is transient weakness, incomplete in degree, passing away in the course of a few hours, but usually recurring after other attacks, and therefore probably of "functional" nature, and a direct result of the effect of the discharge on the motor centres. The second form is that in which paralysis succeeds the first fit, or first series of fits, and persists for weeks or months or permanently, irrespective of the subsequent convulsions. There are cases in which the paralysis is the result of an organic lesion of the brain, of which the convulsions are also the consequence. A third class are those in which persistent paralysis follows a fit in a confirmed epileptic. In these cases, which are rare, a cerebral hæmorrhage has probably resulted from the vascular strain during the fit. More common are cases of convulsions from old organic disease, in which an increase of previous paralytic symptoms follows a severe convulsion. In such cases we may assume that a vessel imperfectly supported or diseased, in a damaged region, has given way during the fit, or that there has been diffuse capillary extravasation.

The rarity of cases of the third class renders them of small practical importance. The second class, "post-hemiplegic convulsions," or "organic epilepsy," will be considered separately. The transient paralysis, which is apt to recur, alone needs consideration among the conditions after attacks.

The motor weakness, which immediately follows a fit, is sometimes distinct when a fit is from the first bilateral. It is then general. The patient may, for some minutes, be unable to walk or even to stand. It rarely, however, lasts long, and is commonly regarded as general exhaustion or prostration from the severity of the convulsion, although it may probably, as Hughlings Jackson has suggested,* be due to the same cause as some of the weakness which succeeds more limited seizures. After convulsions which are partial or unilateral, or which begin unilaterally, motor weakness is both more conspicuous and actually greater than after general convulsions. It usually affects most the limb in which the convulsion begins, and, as the commencement is much more frequent in the arm than in the leg, the weakness is usually greatest in the arm. It is the "epileptic hemiplegia" of Todd, who gave an excellent description of it.†

The duration of the weakness is not considerable, a few hours, or rarely a day or two. The degree varies; it is in most cases incomplete, slight power of movement remains.

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* 'Brain,' April, 1881, p. 439. An elaborate study of the "Exhaustion Paralysis" has been published by Dr. Pierce Clark, 'Arch. of Neurol. and Psychopath.,' New York, vol. ii, 1899.

† 'Clinical Lectures,' edited by Beale, 1861, p. 790.

This is almost always the case in the leg. In the arm, however, the paralysis may be at first absolute, but power returns in the course of a few hours. The same weakness may be left by every fit. If the convulsions recur at short intervals, say every few hours, the paralysis may persist until the series of attacks is over. Sometimes a relation can be traced between the degree of convulsion and the degree of subsequent paralysis, but this is not always the case. In the class of fits which are most frequently succeeded by weakness, those which commence in the hand or the foot, paralysis often succeeds an attack which is extremely slight, and even one of sensory nature, in which convulsion may be absent.

Aphasic loss of speech is sometimes a sequel to right-sided convulsions, especially to those that begin in the face, but sometimes it succeeds fits beginning in the arm or even in the leg, when severe. Such attacks may follow an organic lesion which has prolonged loss of speech for its early effect, and, after each attack, speech may be imperfect for some hours. Such a form of epileptoid aphasia must be distinguished from the inability to speak, which often attends minor attacks, during the momentary impairment of consciousness. There is then often merely unintelligible "mumbling" for a few seconds. Sometimes what is said may be understood, though the patient may be unable to speak distinctly, or that which is said may be unintelligible, although any action is continued.

It will be convenient to consider here the probable nature of the paralysis which succeeds epileptoid seizures, but before doing so certain other conditions found in the limbs after fits may be described. A severe attack leaves the muscles flabby and apparently atonic, and during this condition, in some cases, when the fit has been very severe, the knee-jerk cannot be obtained for a few minutes. The muscle-reflex excitability or "myotatic irritability" may be lost, for a time, apparently from the exhaustion of the spinal centres by their intense activity. More frequently these myotatic contractions are found to be excessive after a fit, so that the foot-clonus can be obtained, although usually only during the first few minutes. This was first pointed out by Hughlings

Jackson* in a case of unilateral convulsion, probably due to organic brain disease, but the foot-clonus is usually to be obtained for a few minutes after all severe epileptic and epileptiform fits. There seems then to be exhaustion of the cerebral centre, leaving the spinal centre uncontrolled and over-active. There may thus be three conditions of these phenomena. After slight attacks there may be no change in the myotatic contractions; after fits of greater severity there may be foot-clonus and increased knee-jerk; and after very severe fits there may be, for a very short time, a loss of the knee-jerk, which is followed by a stage of excess. The relation of the excess to severity of fit is clearly shown by the fact (ascertained by Dr. Beevor) that after a general convulsion, in which each side is apparently affected with equal severity, but in which the deviation of the head shows that the convulsion is really more severe on one side, the clonus can often be obtained on the side to which the head deviates, and not on the other.

Cutaneous reflex action in the limbs (*e. g.* from the sole of the foot) is also abolished for five or ten minutes after a severe fit.

In discussing in this place, for the sake of convenience, the probable pathology of the weakness after convulsions, it must be assumed, in anticipation of the chapter on "Pathology," that the discharge which constitutes the fit is that of cerebral motor centres.

Todd taught that the "epileptic hemiplegia" was due to exhaustion of part of the brain by the excessive action, and the same theory has been accepted by Hughlings Jackson and by A. Robertson, of Glasgow. The former has suggested† that the weakness and foot-clonus may be the result of exhaustion of the fibres of the internal capsule of the brain or lateral columns of the cord by the "discharge" through them, since in organic disease the occurrence of this symptom is especially related to sclerosis of these columns. The hypothesis affords a very probable explanation of some of the weakness after very severe fits, and of the excess of myotatic irritability in these cases; and the loss of the knee-jerk, which is found immediately after some fits, may be due to the initial exhaustion of the reflex centres of the cord. But the exhaustion of the grey substance, in which nerve impulses arise, seems more probable than that of the fibres. An intense excitation of the fibres by electricity causes only a slight diminution in their power of conduction.

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* 'Med. Times and Gazette,' Feb. 12, 1881.

† 'Brain,' April, 1881.

Moreover it is not easy to explain on this theory all the phenomena of post-epileptic paralysis. It is difficult to explain as an effect of "exhaustion" the extreme weakness which may exist in the arm, and occasionally in the leg, after a very slight local fit. There is a disproportion between the severity of the fit and the subsequent paralysis. A patient may have a severe general convulsion, and, a few minutes later, may have no conspicuous weakness; while another patient, who has had a fit confined to one arm, apparently slight in degree, may be unable to move the limb for an hour or more. Since the point is of considerable importance, it may be well to mention the facts of some cases in illustration.

A lad, aged 20, had suffered for ten years from seizures of the following character. Each commenced with a pain in the front of the right shoulder, which passed down the arm "like a knife cutting it," but without any spasm. Then twitching occurred at the angle of the mouth on the right side, the tongue seemed to swell, and he mumbled and did not speak intelligibly. This lasted for a few minutes, and then the attack was over. When the spasm in the face commenced, the pain in the arm always ceased. As soon as the pain commenced in the shoulder, the arm became so weak that he could scarcely move it, and the weakness continued for about a quarter of an hour.

Another patient, a young man aged 25, had had right-sided convulsive fits since measles at six years of age. He presented no sign or history of hemiplegia. Besides the attacks of convulsion he also suffered from minor seizures characterised by sudden "mumbling," the use of wrong words, and weakness of the right arm, without any convulsion.

Again, a woman aged 40, probably the subject of organic disease, having slight right-sided weakness and hemianæsthesia, has also attacks of the following character. They begin with "ticking in the right ear," and this is followed by a painful sensation "like hot needles running into the skin," which passes down the side to the leg and foot, and, after reaching the toes, it returns up the leg and side, and is felt in the arm, hand, and tongue. She mumbles and cannot speak for half an hour. There is no motor spasm, but as soon as the sensation is felt in the leg and arm, the side becomes extremely weak, so that, although ordinarily able to walk fairly well, she becomes unable to stand and scarcely able to raise the arm.

In a patient of 48, with fits since infancy, possibly set up by a small organic lesion, the attacks consisted in "pins and needles" in the left thumb, which passed up the arm to the shoulder and left scapula, with a sense of contraction in the arm, which became powerless, so that she dropped anything that was in the hand.

Several other cases in which sudden loss of power constituted the minor seizures will be mentioned in the chapter in which these attacks are described.

In these cases, then, we have transient paralysis without any motor spasm, *i. e.* we have lowered activity of the motor centres, but without any discharge in them. Usually there is evidence of discharge in the related sensory centres. We must regard the motor centres as restrained or inhibited, and it is readily conceivable that a discharge in the related sensory centre should, under certain circumstances, cause such inhibition, just as a painful cutaneous impression will often inhibit or arrest reflex action, a fact well known to physiologists. If such paralysis accompanying a sensory aura is to be thus, and thus only, explained, it becomes most probable that the weakness which succeeds a slight attack of local spasm is, in part at least, of the same character.

Another interesting fact, to which Hughlings Jackson has called attention, also affords strong evidence that inhibition, rather than exhaustion, is the chief element in the paralysis after local seizures. It is that the paralysis may be greater if a commencing fit is arrested by the ligature, than if it runs its usual course. A pertinent illustration of the same influence has been recorded by Buzzard. Attacks beginning in one hand were succeeded by weakness of the arm for several days. A blister round the arm, during the weakness, restored strength to the arm, but transferred the weakness to the opposite side. The ligature must be regarded as acting on the sensory centre and increasing the resistance in, *i. e.* inhibiting, the related motor centre. That the subsequent paralysis should be thus increased affords strong confirmation of the view that it depends chiefly upon inhibition, while it is incompatible with the theory that it depends solely on exhaustion. The latter, after very severe fits, may, and doubtless does, cause some weakness, probably, however, of very transient duration.

The phenomena of some attacks show that, although the conditions of discharge and inhibition are antagonistic and, in high degrees, incompatible and mutually exclusive, yet in partial degrees they may co-exist. A centre may be in a state of partial inhibition and partial discharge, and either condition may preponderate and ultimately obtain exclusively. It is no doubt difficult to understand the mechanism of such conditions, but we can explain in no other way such cases as that of one patient who, in his attacks, after very slight and transient spasm of the hand, felt as if the arm were being raised above the head in violent spasm, while it was really hanging powerless by his side. Still more striking phenomena of the same kind are those presented by some cases of visual aura, for which this explanation of combined inhibition and discharge has been already suggested. Many of the inhibitory phenomena of the nervous system, *e. g.* of reflex centres, show that antagonistic abnormal states may co-exist in incomplete degree.

Whatever be the exact nature of the temporary paralysis after convulsion, it is noteworthy that the transient condition of the limbs is precisely similar to the permanent condition in cerebral paralysis. There is motor weakness, lowered cutaneous reflex action, excess of the myotatic contractions (tendon-reflexes). When unilateral convulsions occur with great frequency, so that recovery from the weakness caused by one does not occur before another convulsion comes on, a condition of persistent absolute hemiplegia may result, having all the characters of that due to a destructive brain lesion. In a case lately under my care a patient had on three occasions, for about ten days, more than a hundred unilateral fits daily, and complete hemiplegia with inability to speak. On the cessation of each series of fits the hemiplegia passed completely away.

Mental Disturbance.—The mental state usually becomes normal only after a period of sleep. Without, or before, sleep there is usually a slow return of normal condition, the patient being more or less "dazed," speaking at random, and not recognising his surroundings. Transitional states are observed to the automatism to be immediately described. Occasionally there is a more deliberate return of the cerebral activity. One patient, after each fit, was unable to see, and distressing fancies, like nightmare, passed through the mind, and a vague impression that she heard persons talking. Then the sight suddenly returned, and she was perfectly well.

Sensory symptoms, as a sequel to attacks, are not common. One patient, for a short time after waking from the sleep after a fit, heard bells ringing. This was also the prodroma of a fit, for a day or two. There was evidence of slight labyrinthine changes.

Automatic and Hysteroid Phenomena.—After epileptic fits of moderate severity the patient may pass into a condition of mental automatism, in which various acts are performed in an apparently conscious manner, but of which no recollection is afterwards retained. In other cases a patient, after recovery from the epileptic fit, passes into a state of hysteroid convulsion. Both these post-epileptic conditions may occur after major attacks, but scarcely ever when these are very severe. In the description of the minor seizures the automatic actions will be more fully considered, and an account of the post-epileptic hysteroid phenomena is better postponed until the attacks of this character are specially described.

Volition and Convulsion.—An interesting problem is sometimes, though rarely, presented by attacks of slight degree, although not so slight as to bring them into the "minor" category. It is the fact that volitional processes sometimes seem to occur in the centres which are the seat of involuntary discharge. The phenomenon is closely allied to "automatism," as the following instance shows. Just as automatic activity may occur in the uncontrolled centres, after a discharge is over, it is intelligible that the relation of the functional state to the controlling influence should be such as to permit spontaneous ordered action to coincide with slight discharge. A man of nineteen, whose sister was epileptic, had very slight minor seizures from infancy until seventeen, when convulsive attacks began and continued. There was characteristic epileptic clonic spasm in the face and arms, but he was able to go on walking during the attack, and even to cross Oxford Street securely amid the traffic, during the convulsion in the face and arms, but of what he did then he had no subsequent recollection.

Turning on the Face.—An occasional and very grave feature of epileptic attacks is a tendency to turn over on the face at the end of the fit. It seems to be rather a last feature of the convulsion than an automatic action, but its precise nature is not easy to determine. Its gravity arises from the fact that it is common in nocturnal attacks, when it brings the mouth in contact with the pillow and interferes with respiration. The resulting asphyxia deepens the profound post-epileptic coma, and the patient may die. Not only is this the cause of death of many known epileptics, but persons are thus occasionally found dead, with the face against the pillow, who were not known to be epileptic, but had doubtless been the subjects of nocturnal fits, which may go on for many years unknown to the subject of them.

Temperature.—A single fit, although severe, may cause no elevation of the temperature. Sometimes the temperature is raised half a degree or a degree above the interparoxysmal temperature. But when a series of severe general fits occur, separated by short intervals, the temperature may be raised several degrees. This is seen in the "status epilepticus," in which severe convulsions rapidly succeed one another and are attended with considerable elevation of temperature. According to Bourneville, who has carefully studied these cases, in the severer forms of the status epilepticus, which usually end in death, the temperature may rise to 105° or 107°.

Extravasations.—The severity of the strain on the vascular system during a severe fit may lead to the rupture of small vessels and effusion of blood. It occurs most readily in the conjunctiva, where effusions of blood from this cause are not infrequent. It is remarkable that the vessels within the eye seem never to give way. In cases in which there has been a subconjunctival extravasation, I have repeatedly looked for retinal hæmorrhage, but always without result. The support of the encasing sclerotic seems to save the vessels from the effect of the increased pressure. Occasionally minute punctiform extravasations occur on the face, and rarely on other parts of the body. The greater tendency to the occurrence of the extravasations on the face may perhaps be due to the constriction of the turgid neck by the clothes, multiplying the effect of the mechanical congestion. Little is known of the occurrence of internal hæmorrhages during epileptic fits. After death from intense convulsions they are not infrequent in the pericardium, the surface of the brain, and outside the membranes of the spinal cord; but symptoms indicating their occurrence in convulsions which have not proved fatal are extremely rare.

Accidental injuries of various kinds may be received in the fall. The direct effects of the muscular contractions, chiefly dislocations, have been already referred to. Burns, from falling on the fire, are an occasional accident, but are seldom so severe as to be fatal. Cuts on the face or head are common. One patient struck the eye and dislocated the crystalline lens.

Vomiting occasionally occurs after fits, but is not common. It is, however, a dangerous symptom, since the food is apt to get into the glottis. For instance, a man aged 51, who had had many severe fits for three years, had an attack one day, twenty minutes after dinner; when the fit was over, he vomited and died almost immediately, evidently from asphyxia thus caused. Hence inquiry should be made for the symptom, and, if it occurs, the friends should be advised to let the patient lie on the side, that any food brought up should not fall back into the glottis. It varies considerably in character. It may occur immediately, or only some hours later, and is then preceded by nausea. It may occur as soon as the patient wakes from the post-epileptic sleep, or only when prematurely roused from sleep. Sometimes it only follows a series of fits, and not an isolated attack. Very rarely it follows a minor attack, in which there is merely momentary unconsciousness. In all the cases that have come under my own notice the attacks have been those of idiopathic epilepsy. The warning, or the character of the minor attacks if there is no warning, suggests a special relation of the process to the pneumogastric. In one case the aura was a characteristic "globus," in another the minor attacks consisted of this, with nausea and a flow of saliva, or of a pain at the epigastrium. Vomiting after a fit is not related to vertigo or to special sense auras, although a case has been mentioned in which vomiting before a fit was associated with vertigo.

A curious inhibitory relation to vomiting was presented by a patient who has had complete loss of sight after recovering consciousness after a fit until vomiting occurred, in the course of a few minutes. The convulsion was bilateral, and there was a family history of insanity, so that the affection was certainly idiopathic. The feature is of interest in connection with the cessation of migrainous headaches when vomiting occurs.

Another symptom referable to the stomach, which is sometimes met with after fits, is extreme hunger. It is especially common in lads. The patient eats voraciously when he has recovered from an attack, although he may have had a full meal an hour before.

Urine.—The urinary secretion is rarely altered. Occasionally a trace of albumen is to be found in the urine first passed, and, it is said, in extremely rare cases, a trace of sugar. The frequency with which albumen is present has certainly been greatly exaggerated. Huppert* has stated that its occurrence is almost invariable, and that hyaline casts can frequently be found, but most subsequent investigators have failed to corroborate his assertion. Of a large number of cases in which the urine has been examined at the National Hospital for the Paralysed and Epileptic by myself or the several resident medical officers, it has been very rarely that the slightest trace of albumen could be detected by the most careful examination, and in no instance has there been any sugar that could be detected by Fehling's test. I found, however, in a case in which there was organic kidney disease, that the amount of albumen was distinctly increased after the fits. Dr. Beevor examined for me the urine after forty-two attacks in twenty-three patients. In only one instance did he find a trace of albumen, and in this case, after another attack, none could be found.

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* 'Archiv für Psychiatrie,' 1877, p. 189.

The amount of urea excreted was found to be increased after a fit by Echeverria,* but Gibson† found "no constant change in the urine." That the quantity of urea is not necessarily altered is clearly shown by the following estimate which I made of the daily excretion in a case in which very frequent and severe fits occurred. The observations were made on consecutive days.

Number of fits per day.	Urea in grammes.
None	20
One	23·6
Two	18
Several	13
Three	14
Ten	14·5
Five	13·75
Two	13·8

Body-weight.—It has been stated by Kowalewski‡ that every epileptic fit causes a loss of weight, which may vary from one to twelve pounds, and is greater the more recent the disease and the more severe the attack. This is incorrect. It is only in the *status epilepticus* that there is a definite loss of weight, which may amount to fifteen pounds, even when the individual attacks are not severe. In other cases of recurring attacks, even at a few hours' interval, there is also a considerable loss of weight, which is, however, rapidly regained at the end of the series of attacks.

Fields of Vision.—After a severe fit it is not uncommon to find, for a few hours, a concentric limitation of the fields of vision, apparently due to the general exhaustion of the brain, manifested in a function in which it can be readily ascertained. It is not confined, as has been thought,§ to cases with post-epileptic delirium.

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[*] Loc. cit.

[†] 'Med.-Chir. Trans.,' 1867, p. 75.

[‡] 'Archiv für Psychiatrie,' 1881, Bd. xi, Heft 2.

[§] Thomsen, 'Neur. Cent.,' 1883 and 1884.

CHAPTER IV.

SYMPTOMS CONTINUED.

*MINOR ATTACKS.**

THE minor attacks of epilepsy vary much in character, and are generally altogether different from severe attacks; hence patients speak of them under various designations, such as "sensations," "faints," "losses," "turns," "giddiness" and, in America, "spells." Hence also their epileptic nature, and their connection with more severe attacks, are often not suspected by the patients or their friends. It is not always easy to ascertain their exact character, on account of their very short duration, their various forms, and the slight notice taken of them. Indeed, their occurrence is often not noted. It can usually be ascertained from the patient when the onset is attended by a sensation, but when it is not so attended it can only be ascertained from the friends. If the patient is unconscious during the attacks, and there is no warning, he may be absolutely unaware of their occurrence. In many instances consciousness is retained, although it is often modified in some degree, and usually then memory retains traces of its disturbance that can be recalled. But so little note is taken of these slighter attacks, that it is generally necessary to put leading questions regarding the more common subjective disturbances to ascertain whether they occur.

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* In the following account of the subjective sensations of minor attacks, it has been impossible to avoid some repetition of previous statements regarding warnings in general. These have, moreover, necessarily included many facts relating to minor seizures.

The slight attacks met with in "organic epilepsy" in which there is an actual lesion of the brain, usually stationary, differ from those in the idiopathic disease, now to be described, and will be afterwards considered.

Minor attacks of epilepsy may occur alone, even for years, or in association with severe seizures, frequently following these for two or three days, less often preceding them.

The most typical form is transient loss of consciousness without conspicuous convulsion. A patient suddenly stops for a moment in whatever he or she is doing, may then drop what is in the hand, sometimes fall, and then is better.

The abruptness of the loss of consciousness, and also of its return, may be such that neither leaves any trace on the memory, and the patient is only aware of such an attack by some accidental indication of it. Cessation of utterance may reveal it to others to whom he is speaking at the moment. Muscular relaxation is common, partial or complete, in consequence of which the patient may drop what is in his hand, or may fall, and only thus discover what has occurred. There may, indeed, be a return of motor power before consciousness, enough, for instance, to enable the patient to get up after a fall. A gentleman who was dressing by the side of his bath suddenly found himself thoroughly wet. In a slight minor attack he had fallen into the bath, and motor power had returned so as to enable him automatically to get out, and only then had consciousness returned. This is an illustration of automatic action succeeding the seizure, but occurring before consciousness returns, an important feature of many cases, already referred to, and to be considered afterwards in greater detail.

When the onset of such an attack is quite sudden, even a gradual return of consciousness may not permit a distinct recollection of the attack. A young man during a consultation suddenly ceased to speak, and on my repeating a question, "How do you sleep?" replied, "Oh, what is it about?" and for about thirty seconds he replied only by irrelevant questions or remarks. Then his answers had some relation to what was said to him, but for two or three minutes he continued "confused." He was astonished when I told him of his attack, and only recalled the slight final confusion, which, like the memory of a dream, would doubtless soon have vanished. There was no change whatever in aspect throughout the attack. I heard afterwards that he had many similar seizures of which he was unaware.

In such attacks the return of perfect consciousness is frequently as sudden as its loss. A patient may stop speaking, and after a second or two may continue the sentence he had commenced, or he may only discover that he has had an attack by missing something that was in his hand, and finding it on the floor, or behind him if he was walking. It is more common, however, to have a gradual recovery, with confused remarks, such as have been mentioned, which may be of an abrupt character, verging on insolence. This stage, when it cannot be recovered by recollection, may yet leave some trace on memory. For example, a patient, who had had such a brief attack in the street the day before, passed a man, a stranger to him, whose face nevertheless he seemed to know. He was, however, surprised when the stranger said, "I hope you are better, sir." The man had saved the patient from a fall in the attack, but the only effect on memory was a slight sense of familiarity.

It is very common, however, for such attacks to involve only impairment of consciousness, a sense of mental confusion, not actual loss, and for the impairment to be attended by some sensation which can afterwards be recalled. Hence such seizures are often termed "sensations" by those who experience them. Sometimes the sensation is followed by a moment's complete unconsciousness, perceived by the friends, although not known to the patient, to whom only the warning sensation leaves a trace in memory. The sensation may or may not be the same as precedes severe fits, if they occur. Some patients have various forms of minor attacks, attended by different sensations; in others the feeling experienced is always the same. Such indications of minor attacks are very important, because their nature, as already stated, is often unsuspected. They may be frequent when severe attacks are rare, may continue after these have ceased, or precede them for years; thus neither the patient nor the friends have any idea of their nature or significance, and hence they are unmentioned unless specially inquired for.

One of the most common sensory expressions of a minor attack is giddiness, a sense of subjective or objective movement in the patient or the things at which he is looking. He may seem to be turning to right or left, to fall sideways or backwards, or the ground may seem to rise up to him, or he may seem sinking downwards. It is more common, however, for objects to seem to move for a moment, to oscillate from side to side, or move round and round in a circle or an oval in the direction of the hands of a clock. Such sensations are similar to those mentioned as the warning of severe attacks, but subjective vertigo in minor seizures seldom proceeds to the degree of actual rotation, as it often does before severe attacks. Such rotation is the prelude to more energetic cerebral discharges. Occasionally, however, there is an actual movement of turning. Such vertigo always involves, of necessity, an impairment of the correct perception of the relation of the individual to the environment, which is essential to perfect consciousness. This fact seems to be intuitively recognised, so that any sense of impaired consciousness is apt to be described by the patient as "giddiness," and the sensations thus designated are very various. Often on close questioning there is evidently no true vertigo, but only brief obscuration of consciousness, a momentary "dazed" feeling, ceasing suddenly. Hence it is necessary to ascertain precisely what is meant by the word when it is used to describe the sensation.

There is often a remembered sensation of a peculiar mental state. One that is very common is a sudden feeling of "strangeness." The patient may be in a familiar room, but it suddenly seems unfamiliar, or pictures on the walls may seem in some way different. Even when well defined, this momentary "sensation" is often unmentioned until asked for, either because so brief in duration, or else because it has occurred for so long a time, and the patient has become so accustomed to it, that it is [illegible] unimportant.

A sudden brief emot [illegible] scious feature of such attacks, usually a sudden sense of fear. It may precede loss of consciousness, or seem to constitute the whole momentary seizure. Sometimes there is an imaginary cause of the fear, which the patient remembers afterwards, and is more likely the result of the emotion than its cause. In one patient the sudden fear was always accompanied by the idea that a man was running after her. She lost consciousness for a moment, and then was better. She always remembered the illusion afterwards, and recognised its character, but at the moment of its subsequent recurrence it seemed as real as at first. Such a sense of fear may be translated into motion to escape. A girl, subject to similar minor attacks, was speaking to me quite properly, when she suddenly gave a slight frightened scream, and ran down the ward of the hospital. Then she stopped and came back in a natural way, having no memory of what had occurred. The sensation of fear, as a feature of minor attacks, is an illustration of what may be regarded as the fundamental "emotion," aptly so termed, having as its effect an attempt to escape from danger,—the essential condition for the survival of all those whose reliance is on agility rather than strength.

Another mental state that is often described in these attacks is a curious "dreamy" state, deliberate to the patient, although the attack may be very brief to an observer. It is often a pleasant sensation, very seldom definitely disagreeable. Its most frequent form is a sense of reminiscence. It seems to the patient that what is being done, or is around, had been done, or met with, long ago and long forgotten. It is often strangely vivid. Less commonly there is an opposite feeling, that what is being done, and is really quite familiar, has never been done before. Occasionally the sensation is described as the recognition in a dream of some preceding dream, actual or fancied. During the dreams of health a like sensation is sometimes experienced, an incident of a past dream seems to return, and, apparently through its repetition, can be afterwards recalled.

Such a seemingly deliberate mental process is frequently described as a "dreamy" state. It is sometimes associated with some special sense aura, and its occasional association with smacking of the lips, as if from a sensation of taste, seldom remembered, has been the subject of particular discussion by Hughlings Jackson.* The association has been met with in cases of organic disease, especially in the region of the uncinate gyrus, in which there is much evidence to localise the sense of smell.

Special Sensations.—Impressions related to the special senses are common in minor attacks. Visual sensations are especially frequent; the momentary appearance of stars, or a flash of light, sometimes coloured, is common. Large luminous balls, apparently coming nearer, often described as the aura of severe fits, are hardly ever met with in the slight seizures. Sudden darkness, or dimness of sight, is more common, and is often followed by a moment's definite unconsciousness. The scanty evidence organic disease affords regarding the seat of the discharge in such cases, points to the posterior region of the cortex.

Auditory sensations are also frequent, whether or not they constitute the warnings of the severe attacks of the same patient. There is for a moment some sound referred to the ears or to the head, either a hissing or a booming sound, or a more elaborate sensation, as of bells. The sensation may be followed by brief loss of consciousness, or be attended by only obscuration. Especially common is a sense of coincident deafness, passing off with or soon after the sound. It is sometimes a conscious inability to discern the nature of a sound which is distinctly heard, or is a sense that the sounds are far away. The sound heard is sometimes a distinct musical note, and an attack may be induced by the vibrations of the same note. In one case, in which a subjective low note attended the onset of both severe and slight attacks, the latter (the subjective sound with obscuration of consciousness) could always be induced by listening to the lower "A" of the open diapason of the pedal of an organ, one of the low notes of which the vibrations can be felt as well as heard. No other note had the same effect. In almost all cases the sound which attends the slight attacks is of low elaboration—hissing, booming, or a musical note, and is usually referred to the head, or to the exterior, very seldom to the ear itself, as in ordinary tinnitus. More complex sensations of hearing only prelude severe attacks, and are very rare. Dimness of sight may be attended by corresponding failure of hearing; when objects seem small and far away sounds may seem faint and distant.

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* See especially 'Brain,' part lxxxviii, 1899, p. 534, by Hughlings Jackson and Purves Stewart, in which will be found references to several previous papers on the subject by Hughlings Jackson, all well worthy of careful study. Dr. Jackson proposes for the attacks, with symptoms of the perception of a flavour, the name "uncinate convulsions;" but we are only justified in referring the process to this region when there is evidence of a definite olfactory sensation.

Gustatory and Olfactory Sensations.—Pure sensations of taste are hardly ever met with. Those which are so described are always of "flavour," that is, an olfactory sensation combined with some sensation of taste. These are always distinguished by patients from pure olfactory sensations. The latter, as is well known, are due to that which enters the nose by the anterior nares; "flavours" are due to olfactory stimulation through the posterior nares, almost always combined with some true sensation of taste. Simple loss of smell abolishes all perception of flavours. This constant discrimination in epilepsy indicates that there is a central distinction of the two forms of olfactory stimulation maintained in the discharges of epilepsy. I have no note of any case in which a simple sensation of sweetness, sourness, or bitterness was described. When a flavour has been mentioned (called a "taste") it has usually been unpleasant. The sensation is sometimes attended by the smacking of the lips before mentioned, of which the patient is unconscious. But smacking of the lips is much more common apart from any remembered sensations of taste, and frequently occurs in association with the peculiar "dreamy" mental state which has been already referred to. In one case in which a dreamy state, distinctly pleasant, was remembered, there was always the action of spitting; but of this, or of any sensation of "taste," the patient was always unaware. The dreamy state evidently precedes complete loss of consciousness in many cases.

Olfactory sensations, purely such, are not common. A brief sense of an odour may be followed by a moment's loss of consciousness, or more often by merely mental confusion, and then the attack is over. The smell is generally unpleasant in character, as before severe attacks, and is often compared to some decomposing or offensive substance. In one case, clearly epileptic, it was compared to the odour of bad meat burning, but it was found that such destruction of putrid meat was part of the patient's occupation. It is often likened to the smell of bad drains. Very rarely it is described as agreeable, and in such cases it is usually associated with other special sensation. An instance of this is the elaborate and complex special sense aura described on p. 78.

General and Trunk Sensations.—Some form of somatic sensation is a not uncommon accompaniment of minor attacks, but the varieties of this are extremely numerous. An epigastric aura is much less common than before severe fits. A sudden "rushing" sensation is sometimes described, passing from all parts up to the head; it is often spoken of as a "rush of blood" to the head, but is evidently a mere sensation without vascular disturbance; when it reaches the head there is a moment's loss or obscuration of consciousness. Sometimes an ascending sensation is referred to the back only. Other occasional sensations are palpitation and cardiac distress, or a sense of constriction at the throat. In one case a feeling of intense weight at the cardiac region seemed to pass upward, and was followed by a sense of misery in consequence of having done something wrong, ceasing quite suddenly. General tingling or throbbing is sometimes described. But the extreme variety of such sensations baffles even enumeration.

Sudden starts or jerks constitute a not uncommon form of *petit mal.* They may be attended by loss of consciousness, but sometimes the start occurs without its impairment. For instance, one patient who had occasional epileptic fits was liable to these starts every morning, soon after getting up. Both arms jerked suddenly, and apparently the legs also, since, if she were standing at the time, she would fall. Occasionally such a start will throw the patient forward on the knees. These starts are especially common before and after severe attacks. The start may be followed by transient mental dulness, though there is no actual loss of consciousness, as in one patient whose major attacks were severe, and who had minor attacks, one of which I witnessed. She gave a sudden start, put her hand to her head as if in pain, seemed to have a difficulty in speaking for a moment, and then was better. There did not appear to be a moment of actual unconsciousness. That such starts may be truly epileptic is also shown by the fact that very brief tonic spasm may commence by a sudden start—backwards in one patient, who almost instantly began to fidget in automatic action, taking off rings, undoing clothes, &c., and did so for ten minutes—the whole being a blank to memory afterwards. Such starts have no connection with those which occur in fairly normal persons during the process of going to sleep.

Closely allied to the starts is a sudden backward movement. One patient, a girl, had had for fifteen years many attacks every day, in which she suddenly took a step or two backward, looking strange for a moment, and immediately was well. She never fell unless there was some object behind her to trip her up. There was complete unconsciousness for the moment. She was never aware of the attack, except by finding that persons near were looking at her in astonishment. I witnessed many of these seizures, and could never discern the slightest change of colour in the face, which was fairly ruddy. Another patient, in the minor attacks, always suddenly went back, although his face was drawn slightly to the right; consciousness was lost for a moment, and then he was better. Allied to starts is a sudden brief loss of power in the legs, which also may cause a fall.

Urine may be passed in minor attacks of any character in which there is loss of consciousness, but micturition is far less common than in the major fits. It is more frequent in sleep-attacks than in those that occur in the day. As already mentioned, urine may sometimes be passed when the patient is not completely unconscious, as in one patient whose minor attacks consisted in sudden giddiness (objects going to the left), and a feeling of fear. She always passed urine in the attack, and knew when she did so, but could not prevent it. Sometimes it occurs when the unconsciousness is so brief as to elude the observation of the patient or of those about him.

In the cases of organic epilepsy, in which the fits begin locally in one limb, the minor attacks usually consist solely in the spasm, or sensation, with which the major attacks commence. When limited to the limb there is seldom loss of consciousness. Sometimes there is local spasm of this kind in cases in which the severer fits are so rapid in their development that the convulsion is apparently at once universal, and loss of consciousness is so early that there is no warning. But one patient, for instance, who had such severe epileptic fits, had also slight attacks in which, as in one that I witnessed, she suddenly began looking round and mumbling, without the least change of colour, then lay back in the chair in which she was sitting; the right arm was stretched out, then became stiff, with the fingers flexed; respiration was difficult for a moment, and the pupils became larger. Then the attack was over; but she seemed stupid, and could not be made to speak for a few minutes.

In some of these cases the spasm may be variable in its seat. A man had at first very slight attacks, consisting of sudden rigidity of some part of his body, rendering it immoveable, sometimes one limb, sometimes the jaw, and each lasting, he conjectured, five or ten minutes. He had also other minor attacks characterised by "dizziness." He subsequently became subject to severe general convulsions.

Another form of minor attack, in patients whose severer fits begin unilaterally (especially those who suffer from "organic epilepsy," the result of an old organic lesion of the brain), consists in sudden weakness of the limb in which the severer attacks commence. For example, a patient who was liable to severe left-sided fits preceded by a throat aura, had also minor attacks which consisted in sudden giddiness and inability to move the left side for a few minutes. Another patient, a man aged 28, wi[illegible]t history of syphilis, had suffered for three yea[illegible]ts which commenced in the right side of the f[illegible] then consciousness was lost, and the right arm and leg were convulsed, and afterwards the left limbs. For a day or two after each fit his speech was altered, and the right arm and leg were weak. He had also minor attacks which commenced by a noise in the throat, and consisted in inability to speak and inability to move the right arm and leg, there being no convulsion. These cases have been already alluded to on account of the evidence they afford that weakness associated with epileptoid seizures may in minor attacks be due to inhibition of the centres which are discharged in severer attacks. This is also seen in patients whose primary disease is in the speech region of the cortex. Attacks may ensue in which brief unconsciousness is followed by loss of speech, sometimes for hours, passing off with misuse of words.

In many of the cases in which the slight attacks are attended by a definite and uniform sensation, the severe fits occur without warning. This is especially the case when the minor fits consist of vertigo only. When the severe attacks are attended by a definite aura, this, as already stated, may or may not correspond to the sensation which attends the minor attacks. For instance, one patient whose severe attacks began with a sensation in the eyeballs as if they were being pushed back into the head, and rotation of the head to the right, had minor attacks consisting in a sudden sensation of sparks before the eyes. Another patient, whose minor attacks consisted in a "sudden smell like sulphur which seemed to stir up from the pit of the stomach," had also severe attacks, of which the warning was a sudden sound. Another patient, whose major attacks were preceded by a sensation of "crawling" on the soles of the feet, which passed up to her head, had also minor attacks which consisted in a sudden sense of extreme fear.

It is, however, much more common, when the major attacks are preceded by a definite warning, for the slight fits to be preceded by, or to consist of, the same sensation. This may be combined with some other warning. A patient whose severe attacks were preceded by a sense of alarm had slighter attacks consisting of alarm combined with giddiness.

Such sensory symptoms may occur, to judge from the objective symptoms that are presented, and yet leave no trace which permits their subsequent "recollection." The smacking of the lips, which is so common, probably results from an unremembered sensation of taste. One patient who had suffered from attacks of momentary cardiac pain and faintness, had afterwards seizures in which her hand was put on the epigastrium with a scream of suffering, but no sensation was afterwards remembered.

The sensations which attend the minor attacks may vary in the same patient at different times. A girl whose severe fits commenced by a burning sensation at the epigastrium and sense of choking, and were distinctly epileptic, attended by tongue-biting, had slighter seizures, which consisted at first in a burning pain at the epigastrium and lower part of the sternum, lasting a few minutes and followed by sickness, but at a subsequent period they consisted in transient loss of sight, followed, as sight was returning, by dazzling sparks of colour, especially red. Another patient would have attacks of momentary weakness, sometimes with, sometimes without, an auditory sensation. In one, loss of consciousness sometimes occurred alone, sometimes preceded by a "rush to the head," in each case followed by automatic action. In a third there was, at different times, a sense of strangeness, momentary vertigo, sudden loss of sight, star-like lights. In a fourth, giddiness, a sense of strangeness, alternated with an epigastric sensation rising to the throat. Examples of such variation might be multiplied indefinitely.

Conditions after Minor Attacks.—In many cases consciousness is obscured for a few minutes after a minor fit; the patient remains "dazed" and stupid, answering questions with difficulty, and sometimes going to sleep. In other cases the patient, during this stage, performs some actions automatically, or passes into a condition of hysteroid convulsion, sometimes with maniacal violence. These post-epileptic phenomena may, as already stated, occur after moderately severe fits, but are more common the slighter the attack, the more severe [illegible] only by deep sleep. The inverse relation of automatic action to severity is sometimes to be observed in the same patient. These after-states are of great importance, and must be considered in detail.

Automatism.—The automatic action is often regarded as constituting the epileptic seizure, and the attacks presenting this feature have, following Esquirol, been called "masked epilepsy," or "epilepsia larvata" after Morel. Whether this mental automatism may replace and represent an epileptic seizure, or whether it is always a post-epileptic phenomenon, is a question still undecided, which will be presently considered.

It is certain, however, that the automatic state often succeeds an attack, and is then essentially a post-epileptic phenomenon. According to an hypothesis suggested by Anstie and Thompson-Dickson, and applied by Hughlings Jackson, it is the result of the exhaustion of the highest cerebral centres by the discharge, and the consequent temporary loss of the control which these should exercise over complex centres functionally below them, which consequently act in an insubordinate and automatic manner.

These automatic actions are not merely of clinical interest, but also of practical importance on account of their medico-legal aspect, since they are sometimes complex and have the aspect of voluntary actions. It is, indeed, often not easy to convince observers that these actions are not deliberately volitional and intentional, so apparently conscious are the patients: but consciousness is in an abnormal state, for the memory often retains no recollection of these actions. It is evident that they develop during the stage of slow recovery of consciousness. Sometimes, indeed, there is a sudden perfect return of the normal state, but more often there is a gradual recovery of perception of surroundings and of the character of sensory impressions. The patient may not know where he is, may ask some strange question, or repeat one that is asked him, and slowly become well.

In the simplest form, a patient, after an attack, may seem dull and stupid for a moment, proceed to perform some inapposite action—sometimes suddenly, as if under the influence of emotion, sometimes deliberately—in a dreamy manner. A very common form of deliberate action is that of undressing, and it occasionally has serious practical inconveniences. The act of undressing is perhaps the result of a sensation of illness which suggests going to bed. In one case it accompanied a fall in the pulse-rate from 80 to 35, a symptom which may have been due to a cerebral state causing a sense of illness. Another occasional action which may be the result of the same suggestion, is an attempt to walk up a flight of stairs which the patient thinks is before him. Thus one man, who had his fit in a kitchen, thought that the shelves of a dresser were stairs, and tried to walk up them. Another patient had a slight fit at dinner, and immediately stepped on to the table and made movements as if going upstairs. Usually, as in this instance, the action is a simple and a natural one, only rendered equivocal by the surrounding circumstances. Circumstances may, indeed, render the automatic action still more awkward. A man of 29, with heredity and infantile convulsions, had epileptic fits, slight usually, but sometimes severe, since the age of ten. The slight attacks were characterised by brief loss of consciousness, with slight deviation of the head, and pallor of the face. When its colour returned he began speaking incoherently, beginning one sentence, finishing it with another, and then would undress himself, letting his trousers down. He was a teacher of music, and the equivocal character of his automatic action when teaching a young lady compelled him to give up his profession. Another similar action is to put into the pocket any object that he may be near, irrespective of its ownership. A young man who presented this symptom was a draper's assistant. He had minor attacks which consisted only in brief loss of consciousness, preceded by an olfactory aura—a "nasty, indescribable smell in his nose and mouth, a sort of combination of smell and taste." After the attacks he constantly found in his pocket any object which had been near him when the attack came on, such as [illegible], reels of cotton, &c. This patient had always a sensation of hunger after the attacks, and he occasionally only discovered that he had had a seizure by finding, to his surprise, that he was cutting bread and butter and eating it as fast as he could. It is a not uncommon thing for patients in the hospital, after slight attacks, during the automatic state, to go to other patients' lockers, take things out, and put them in their own pockets. One woman whose general conduct suggested no suspicion of dishonesty, after an attack went to the locker of another patient, took out a purse, and pocketed it.

Automatism follows even the slightest attacks if these are definite in character. In one, seen by me, there was a moment's headache, the patient turned round twice, took up a book and put it in his locker; then he rubbed his eyes and realised that something had happened, but remembered no particulars.

Occasionally the actions performed are extremely complex. I have known, for instance, a carman, after an attack, to drive through the most crowded parts of London for an hour without an accident, and retain no recollection of it afterwards. I have even heard (from Dr. Radcliffe) of a young lady playing, during this state, the most difficult music without mistake. Trousseau relates the case of an architect who, when seized on a scaffold, would run quickly from plank to plank for a few seconds, shouting out his own name. He never fell. The state is no doubt closely allied to the condition in somnambulism, in which the precision of muscular action is well known.

Emotional Automatism.—Sudden automatic action after a fit is often distinctly associated with an emotional state. Occasionally this is intense, and may be manifested by violence. It is probable that most cases of epileptic mania are really examples of post-epileptic automatism. Even the irrelevant answers to questions during the few minutes after a slight minor attack are often uttered in such an insolent and aggressive manner as to suggest some act of violence. A patient liable to minor attacks, stood still in one while crossing a crowded street; a passer-by took him by the arm and led him to the safer pavement. The helper then was astonished to receive a severe blow from the person he had helped, who looked at him with indignation and anger.

Another patient, immediately after an attack, struck a friend who was walking with him a violent blow on the face, mistaking him for another person, and was in consequence taken to a police station under the suspicion of being drunk. One woman, immediately after a fit, threw her baby downstairs. Without doubt, many crimes have been committed in this state. The possible relation of criminal actions to epilepsy has formed a medico-legal question at many criminal trials.

The emotion displayed is sometimes of a more cheerful character. One patient always laughed and sang for a few minutes after every attack of *petit mal*, and in another, a girl of twenty, each slight seizure was followed by an impulse to kiss; she would thus salute every person near her, irrespective of sex, and even seize her pillow and kiss it most affectionately.

In some cases there is considerable uniformity in the character of the actions which are performed in this post-epileptic state. A woman, for instance, aged 45, had been the subject of epilepsy since six years old; severe attacks, with tongue-biting, occurred every few months, and slight attacks, consisting of the epigastric sensation and loss of consciousness, much more frequently. From the first, after both kinds of fits, she commonly tore her hair. When quite a little girl she was once found unconscious in a room by herself, and had torn a quantity of hair out of her head.

It has been already mentioned that automatic action sometimes occurs at the apparent onset of the attack. A patient may fumble with the hands, or perform some more definite action, without any preceding loss of consciousness, so far as can be discerned.

Post-Epileptic Hysteria.—Instead of presenting automatic action of the character described, some patients pass, on the cessation of an epileptic fit, into a state of hysteroid convulsion. This sequel is met with only in patients who are of the age at which hysteria is common—namely, under thirty-five; and although common in both boys and girls, it is comparatively rare in young me [illegible] in you
women. Some patients a [illegible] ttac

others never do. Hence it is evidently the result of two causes. The first is the preceding cerebral "discharge," leading to insubordinate action of the lower centres, perhaps in consequence of the mere loss of control of the highest. Secondly, it is the result of some functional state of the brain, such as leads to the manifestations of hysteria, apart from epilepsy. It is the possession of this special instability which leads to the occurrence of the hysteroid convulsion under the circumstances. Other patients, without this special predisposition, have quite similar attacks, but without any hysteroid sequelæ. In some of the patients who present these symptoms there are, at other times, various manifestations of the hysterical temperament; in others these are absent, except after the epileptic seizures. In the latter the hysterical condition is apparently not sufficiently developed to lead to independent symptoms, although it manifests itself during the morbid stage immediately after an epileptic fit. Hence we must regard the patients who present this sequel as really the subjects of both epilepsy and hysteria. Some of them have only attacks of *petit mal*, and in such cases it may be extremely difficult to say whether the hysteroid attack is simple, exists alone, or whether it is preceded by an attack of minor epilepsy, and is really a post-epileptic phenomenon. On the other hand it is very common for these post-epileptic hysteroid convulsions to be mistaken for major epileptic seizures. Patients are often said to have frequent attacks of *petit mal*, and occasionally severe fits, or to have been liable to slight attacks for months or years, and then to become subject to severe attacks, when the latter are found, on minute inquiry or actual observation, to be merely hysteroid convulsion occurring after an attack of *petit mal*.

The occurrence of such convulsion, as an occasional sequel to severe attacks of epilepsy, has been already mentioned. It is, however, far more frequent after minor seizures. But it will be convenient to postpone the description of these attacks until the character of hysteroid seizures is considered in detail.

Post-epileptic Pronation.—There is one post-epileptic action that is of great importance on account of its danger. It is the tendency to turn over on to the face. When the patient is in bed, suffocation may easily occur in this posture, and without doubt many epileptics have died from this cause. Some patients never present this tendency; others always do, and their friends should be made aware of its danger. The peril is the greater, because the action is especially common in nocturnal fits, minor or moderately severe, and the combination of the soft pillow and solitude constitutes a grave danger. It is increased by the fact that nocturnal fits, in those who are unmarried, may occur through many years unsuspected. I believe that it may be accepted as an axiom that when a person is found dead in bed, with the face against the pillow, death resulted from suffocation in the post-epileptic state, even though epilepsy was not known. Immediately after a fit, asphyxia seems to have no power to arouse the brain, and the stage of lethal unconsciousness seems to be at once attained. Moreover, the tendency may follow brief post-epileptic hysteroid convulsion, and patients who present it are therefore often thought to suffer merely from hysterical fits. It was illustrated, only too clearly, by the case of a girl, aged 15, who had suffered from "fits" for a year. There was strong hereditary tendency. Her own father and brother were insane. Her mother's father and brother were both epileptic and insane. Her fits occurred daily, both sleeping and waking. The history of epileptoid seizures was clear, but after the fit was over she would turn on to her stomach and then kick and struggle, sometimes for hours. In the hospital, however, she had no distinct epileptic attacks, and the attendants were inclined to regard her attacks as purely hysterical. She sometimes fell down on her back and lay quiet without change of colour, and could then be brought to by stopping the mouth and nose for a few seconds. In other attacks she would fall suddenly and then clasp her hands tightly, stretch herself out stiff and "work her eyes about," and always turn on to her face and try to press her face into the pillow or against the floor. From this state she could be quickly roused by water. One night she was noticed to be sleeping quietly at 3 a.m. At six o'clock the nurse went to her bed to call her, and found her lying prone, with the face against the pillow, and dead. No one in the ward had heard a sound.

This case brings before us the nature of the tendency. In some cases there is obtrusive initial vertigo and deviation of the head. In these the turning over occurs immediately, without "purposive" movement. It may well be a residue of the unequal degree of discharge in the two hemispheres which causes the initial vertigo, deviation of the head, and sometimes rotation of the trunk. The persistence of the influence of this inequality is shown by the fact that the foot-clonus lasts longer after the fit on the side towards which the head turned at the onset, and it is conceivable that a residue of this influence may induce the turning over. The special and important relation to nocturnal attacks is, moreover, perhaps simply related to the horizontal posture. A child of thirteen had attacks in the day; after deviation of the head and slight commencing rotation of the body, she fell unconscious. There was little visible spasm. The attack was soon over. But at night, when asleep, the same deviation of the head passed on to rotation of the body, so that the slight attack occurred with the face against the pillow. Here it was clearly not post-epileptic, but the result of the initial process of the fit. The turning over occurs in the horizontal posture only because the process produces a fall in a patient who is standing or sitting.

When this is manifested as part of the hysteroid automatism it is still possible that a remaining influence of the initial vertigo, using the word in its widest sense, may be the determining cause of the movement. It is seldom, if ever, met with when the post-epileptic hysteroid convulsion is of long duration and intense degree,—seldom, that is, when it is such as might be conceived to obliterate all effects of the epileptic discharge.

MENTAL DISTURBANCE IN EPILEPTICS.

Paroxysmal Mental Disturbance; Epileptic Mania.—The subjects of epilepsy sometimes, but rarely, present sudden paroxysmal outbursts of mental derangement, often with violence, and a tendency to injure others. The maniacal attack is usually brief, often lasting for a few minutes only, sometimes for an hour or two. It was formerly thought that such outbursts represented and constituted an attack of epilepsy. In the majority of cases, however, it is certain that, as Hughlings Jackson maintains,[*] the mental derangement is the sequel to a fit, and is really a form of the automatic action which may succeed an attack, sometimes severe, much more commonly slight.

If the preceding minor attack is very slight, it may readily be overlooked, and hence the psychical disturbance be regarded as primary. Whether this is true of all instances of maniacal outbursts in connection with epilepsy is doubtful. In rare cases (as Esquirol and other good observers have pointed out) an act of violence precedes a fit. We have seen, in the account of modes of onset, that the aura of an attack may sometimes consist in a complex or co-ordinated mental process, or a visual idea. There is nothing inconsistent with ascertained facts in a sudden act of violence as the result of the initial discharge. One of my patients always hopped round the room before he fell in a fit. Cases in which a patient suddenly runs, or turns round and walks backwards, before a fit, have been mentioned, and a similar cerebral process to that which causes such a proceeding may declare itself by an act of violence. It seems thus possible that a maniacal outburst may be the only effect of a "discharge," may alone constitute an attack. Such cases are certainly rare, but it is doubtful whether we are justified in denying their occurrence.

Occasionally, after a fit, or, more frequently, after a series of fits, an attack of mental disturbance may come on which lasts for several days. It may be simply a demented state, as there may be hallucinations, with irritability and even violence. This state sometimes comes on in an epileptic patient without immediate association with attacks, which indeed are often absent during the period of temporary mental aberration. There is more tendency to an attack of positive insanity in proportion to the amount of psychical disturbance associated with the attacks. It should also be noted that, apart from such considerable derangement, it is not uncommon for a patient's mental state to be worse, and his temper more irritable, when severe fits are arrested, and this apart from the occurrence of slighter seizures.

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[*] 'Med. Press and Cir [illegible] 1874, p. 409.

Interparoxysmal Mental State.—The mental state of epileptics, as is well known, frequently presents deterioration, and this constitutes one of the consequences of the disease which is much dreaded, and is often most serious. In its slighter form there is merely defective memory, especially for recent acquisitions. In more severe degree there is greater imperfection of intellectual power, weakened capacity for attention, and often defective moral control. Mischievous restlessness and irritability in childhood may develop to vicious and even criminal tendencies in adult life. Every grade of intellectual defect may be met with, down to actual imbecility.

The mental state must not be regarded, in all cases, as entirely the effect of the disease. It is certainly, in some, the expression of a cerebral imperfection of which the epilepsy is another manifestation. In such instances mental defect exists from the onset. In many cases, however, the failure must be regarded as a consequence of the disease. It distinctly succeeds the fits in point of time, and may lessen very much when the fits are arrested by treatment. It is not surprising, therefore, that in cases in which slight mental defect exists before the fits commenced, this should be greatly intensified by the subsequent attacks. It is among these cases that many of the worst forms of mental failure are met with. In cases of long duration in childhood, and especially those that date from quite early life, the mental defect is often stamped upon the features; a constant frown gives a peculiarly ill-tempered look.

The conditions with which the occurrence of mental defect is associated have been carefully compared in the cases in which considerable mental defect existed, and of which the history could be ascertained.

It is doubtful whether *sex* influences its occurrence. There was rather greater preponderance of females than obtained in the whole series, but the difference is scarcely sufficient to be of significance. A comparison of cases seems to show that heredity does not increase the probability of mental failure.

The *age* at which the disease commences has, however, a very distinct influence. The tendency to mental failure is, as Romberg pointed out, greatest in the cases which commence in childhood, under ten years, and is much less in those which commence between twenty and thirty. It is also greater in early than later childhood, the first five years yielding no less than 39 per cent. of these cases, and only 18 per cent. of the whole series. This, doubtless, depends upon two causes. During early life the brain suffers in great degree from any unfavourable influences; and secondly, many children with cerebral defect, congenital, or the result of damage during birth, become epileptic during the first five years of life. Repeated severe fits in a child, if long continued, sometimes seem, by their influence on the development of the brain, to induce a state of imbecility which may be permanent. Mental failure is, however, occasionally met with in cases of epilepsy which commence during the second half of life, and the two are then common results of a degenerative tendency.

The *duration* of the disease has also a distinct influence on the occurrence of mental failure. The disease had existed for many years in a larger proportion of the cases which presented mental failure when they came under observation, than of those which presented no conspicuous failure. Of the cases with mental failure nearly three quarters had lasted more than four years, while less than half the cases without mental failure were of this duration. Nevertheless, cases of very long duration are much more common without than with mental defect. Thus no case with considerable mental defect had lasted for more than twenty-one years, while the series of cases without mental change included many of longer duration than twenty-one years, and some of longer duration than thirty years; in one the disease had existed for fifty-four years. The reason for this probably is that the cases of very long duration with much mental change usually find their way into asylums.

The long duration of some of the cases without mental defect illustrates the fact, that the intellectual change is not a simple effect of the duration of the disease, which is one, but not the only, and probably not the chief, factor in its causation.

Frequency of Attacks.—An examination of the frequency of attacks shows that it cannot be regarded as possessing a considerable influence. The chief difference is the comparative rarity of long intervals in cases with mental failure. In only one of the cases did the interval between the fits exceed two months, whereas in the series without mental defect this interval was often exceeded. The average interval in the latter series (without mental failure) was twenty-six days, whereas in the cases with mental defect it was only fifteen days. But the fact that in the two series the proportion of cases in which the intervals were very short is nearly the same, shows that mere frequency of attack can scarcely be regarded, by itself, as a cause of mental failure.

The influence of *form of attack* on mental failure is a subject on which great diversity of opinion exists. Most authorities are of opinion that minor attacks, in which there is loss of consciousness and little more, exert a more prejudicial influence than the severe fits. It is certain that the gravest forms of mental defect are sometimes met with in cases in which there are only minor attacks, but they also occur in cases in which the attacks are severe and *petit mal* is absent. On the other hand, that minor fits, even when frequent and occurring for a long period, do not necessarily cause mental failure, is certain. One of my patients, a girl of twenty-five, had suffered for twenty years from *petit mal*, with complete loss of consciousness; thirty to forty attacks occurred daily, and yet there was no defect of mental power —not even of memory. In another case, with strong inheritance, forty minor attacks occurred daily, consisting of fixation of the eyes and momentary loss of consciousness, but there was no mental failure. In another the state of mind was quite normal, although a dozen minor seizures had occurred daily for four years. Hence it would seem that no necessary, and perhaps no considerable, influence is exerted by mere form of attack, so far as the distinction between slight and severe fits is concerned, or even by frequency considered alone. That there are varieties of these two forms which exert a more prejudicial influence than others, is highly probable, but evidence of their character is not yet forthcoming.

The conclusion from these considerations is that mental failure is determined less by single conditions than by their combinations, and that it is probable that a more potent cause than the attacks themselves consists in a predisposition to suffer under their influence—a predisposition which is related to the ultimate causes of the disease rather than to its developed characters. Of the latter, early age at commencement, long duration of the disease, and frequency of attack are more influential than the sex of the sufferer, the existence of heredity, or even the character of the attacks, so far as concerns the mere distinction between major and minor fits.

In many cases of senile epilepsy, in which the malady is an expression of degenerative changes in the brain, mental change may be another result, depression, loss of memory, hallucinations, or delusions, which are usually lasting.

GENERAL HEALTH OF EPILEPTICS.

So far as can be ascertained, many epileptic patients are in perfect and even robust health. The various organic functions, in the intervals of the paroxysms, are normal. More frequently, however, the general health exhibits some imperfection. The digestive organs are often deranged, the bowels are constipated, flatulence is troublesome, the appetite bad, and the tongue is furred. In some patients there is a voracious appetite, especially after attacks. Often the disturbance is increased by the long-continued administration of bromides. The circulation is feeble, the pulse small, unduly frequent, and very often slightly irregular. The heart's action is excited, and slight dilatation of the left ventricle can frequently be recognised. "Nervous urine," pale and of low specific gravity, is frequently passed. The constitution of the urine is usually normal. The transient passage of albumen or sugar, after an attack, is so rare as not to deserve mention.

CHAPTER V

ORGANIC EPILEPSY

Many organic diseases of the brain not only cause convulsions during their active stage, but, when their active stage is over, are followed by epileptiform attacks which may recur for many years, sometimes during the whole of life. They are usually partial and local in commencement, but may be general when severe, and often resemble idiopathic epilepsy in another feature—the occurrence after a time of minor attacks identical with those that attend the latter disease. Such convulsions, for instance, are not uncommon as a sequel to hemiplegia at any period of life, especially when the seat of the disease is in the cerebral cortex. In many cases of the same class the initial palsy is inconspicuous or absent, although the circumstances of the onset and the character of the convulsions make it certain that they are due to organic disease. These cases are of considerable practical importance; the tendency to discharge is established by repetition. The ultimate course of the disease resembles that of idiopathic epilepsy, and these cases must be included in any survey of the malady. Many features of local onset described in the foregoing pages are ascertained from cases probably or certainly of this class.

Etiology.—Hemiplegia, occurring at any period of life, may be followed by recurring convulsions; but this sequel is far more frequent in the cases in which the onset of the paralysis is during childhood, than in the cases of hemiplegia in adult life.

In two thirds of the cases the hemiplegia came on under five years of age. In nearly half the cases the onset was before the end of the second year. It seldom actually dates from birth. At all periods of adult life, however, a cerebral lesion may occur which has convulsions for its sequel. Females suffer from post-hemiplegic epilepsy more frequently than males, but this depends especially on the larger number of girls than boys who become affected under five years, the former being nearly twice as numerous as the latter. In the small number of cases in which the hemiplegia comes on after twenty, males preponderate.

It is more common for the hemiplegia, in these cases, to be on the left side than on the right, but this statement is only true of the cases in which the affection comes on in early childhood.

The cause of hemiplegia in these cases can only be inferred from the conditions of its onset. In young children sudden hemiplegia may be the result of spontaneous thrombosis in a cerebral vessel. There is also evidence that it may result from local inflammation of the grey matter, analogous to that which, in the spinal cord, causes the common "infantile paralysis." In many cases no exciting cause can be ascertained. In a few the onset is during an acute illness—during measles, during convalescence from scarlet fever, during mumps, in the subjects of inherited syphilis,—in many cases a fall on the head is the apparent cause. The history of many of the cases which occurred after childhood points to a local lesion. Some are the subjects of constitutional syphilis, in others there is heart disease or the onset was during rheumatic fever. Thus in many cases there was reason to believe that the lesion was due to vascular obstruction. The same opinion has been expressed by Hughlings Jackson.* It is well known that in softening from this cause the brain tissue adjacent to the softened area is usually damaged by the collateral congestion, which leads to abundant punctiform extravasation; if grey matter is thus damaged, it is easy to understand that it may recover imperfectly, and that a permanent condition of instability may result. Moreover, thrombosis in veins (a common lesion in childhood) often affects the surface of the brain, where organic disease most frequently causes convulsions. In other cases the onset is during an acute cerebral illness, possibly meningitis. In some the initial hemiplegia occurs without assignable exciting cause during hot weather, and is commonly ascribed to polio-encephalitis, inflammation of the grey substance of the cortex, analogous to the polio-myelitis which causes atrophic spinal paralysis.

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* 'Brain,' April, 1881, p. 437.

In some of the cases which dated from birth, labour had been difficult, and it is probable that injury to the surface of the brain occurred during parturition. This may be an occasional cause of epilepsy when there is no paralysis. It should be suspected whenever convulsions occur during the first few days after birth, especially in a first child who was born tediously and lived with difficulty.

In most cases no other cause for the convulsions could be traced than damage to the brain. In a few, however, there was an inherited tendency to epilepsy. Two adults were the subjects of lead-poisoning and albuminuria; the former is known to be capable alone of causing convulsions, and may have produced a predisposition to their occurrence.

Symptoms: Onset.—In half the cases convulsions attended the onset of the hemiplegia, which was, in most of these, in early childhood. These initial convulsions were usually severe and repeated (see p. 23). Sometimes two or three attacks occurred, involving one side, and after the last the child was found to be paralysed. In other cases the convulsions recurred during several days, and even a week.

The chronic convulsions which succeed the hemiplegia date from its onset in one half of the cases. A few days or weeks after the establishment of the paralysis the patient has another fit, soon afterwards another, and so on until the convulsive disease is established. A bright boy of two and a quarter had a severe attack of whooping-cough, and, at its worst, right-sided convulsions occurred and were repeated during three days with persistent loss of consciousness. When this returned he had lost his speech and there was complete right hemiplegia. Power returned in a few months, but a year later he could only say "Mother." The fits recurred every few days, and his aspect was that of grave mental defect. An extensive cortical lesion must have occurred, perhaps from venous rupture during asphyxial cough, damaging the regions concerned not only with motion, but with mental processes. Enduring aphasia does not result in early life from a lesion limited to the speech region; it always indicates extensive mental impairment.

In other cases there is an interval after the onset of the paralysis before the recurring convulsions. The patient may be well, except for the remains of the hemiplegia, and after a few months or years may become subject to fits. In about a sixth of the cases the interval is more than five years, and occasionally it is as long as fifteen or twenty years. If the fits do not recur within a week, there is usually an interval of more than a year.

In some of the cases in which an interval elapses, the recurrence of convulsive attacks may be traced to some exciting cause. In one patient, for instance, the hemiplegia came on without apparent cause at one year old; the onset was attended by convulsion, but no other fits occurred until two, during an attack of whooping-cough. In another case the hemiplegia came on without convulsions at four months, but fits occurred during the first dentition, and again during the second dentition at seven, and after the latter they were permanent. Again, one patient had hemiplegia, after a fall on the head, at two years of age, with convulsions at the onset, but no other fits until seven years of age, when the attacks recurred after another fall on the head, and continued until the patient came under treatment at eleven. It is not very rare, in cases in which the hemiplegia comes on during the first years of life, for the patient to be free until puberty, and then, at thirteen, fourteen, fifteen, or sixteen, the fits commence and continue. No doubt, in these cases, their occurrence is due to the influences which determine the frequent commencement of idiopathic epilepsy at this period.

The only relation ascertainable between the occurrence of convulsion at the onset of the hemiplegia and the interval which elapses before subsequent fits, is that, in the cases in which the persistent attacks commence soon after the onset, this is accompanied by convulsion. The converse, however, is not true. In many cases in which convulsion occurred at the onset, an interval of several years elapsed before the fits recurred.

In the cases in which convulsions succeed hemiplegia which comes on late in life, there is rarely any long interval of freedom after the onset of the hemiplegia. Usually, in the course of a few months, or at most of a year, the recurring seizures commence.

The condition of the paralysed limbs in these cases varies. In most cases a considerable degree of paralysis persists, especially in the arm. That in the face is usually slight, and in the tongue often absent. The leg has usually recovered almost completely, as is the rule in hemiplegia occurring in early life. The weakness in the arm is, in most cases, distinct, and often great; sometimes the hand remains completely paralysed, a little power in the shoulder and elbow alone remaining. In other cases recovery is almost perfect; the patient may be unconscious of any persistent weakness, although usually slightly defective power in the arm or face may be found if carefully searched for. In very rare cases no trace can be found of any weakness, although the initial hemiplegia may have been well marked, and the convulsions may affect that side only. When the hemiplegia comes on in early life, the paralysed limbs are usually smaller than those on the other side, and smaller not only in circumference but in length, in consequence of a retardation in the growth of the bones, in which even the scapula may participate. The arm and leg may each be one or two inches shorter than that of the other side. The arm may be the seat of late rigidity. The hand is often affected by a condition of "mobile spasm," which has been called "post-hemiplegic chorea" by Weir Mitchell and Charcot—spasm which causes slow spontaneous movements of the fingers and thumb, and incoordination of voluntary movement. In an account of this form of post-hemiplegic spasm* I have pointed out the frequency with which it is associated with convulsions, and the association is equally conspicuous if these cases are studied from the side of epilepsy. Such spasm is found in one half of the cases of post-hemiplegic epilepsy. Its character and degree vary. The chief feature is the tendency to spasm of the interossei, whereby the fingers become flexed at the metacarpo-phalangeal joints and extended at the middle and distal articulations. Frequently the continued extension at these joints leads to over-extension and subluxation, so that the round heads of the first phalanges project unduly on the palmar aspects of the fingers. This form of spasm presents a striking contrast to that of "late rigidity," in which the spasmodic contraction chiefly affects the long flexor of the fingers, which are thus flexed at the middle and distal joints. The thumb may be bent in to the palm, or pressed against the first finger, or over-extended. Often the wrist is strongly flexed by the contraction of the special carpal flexors. In other cases, or at other times, the wrist becomes strongly extended. When the mobile spasm is slow, considerable, and spontaneous, the hand presents the condition known as "athetosis." In other cases the spasm may be slight and inconspicuous when the hand is at rest, but may be distinct on movement, causing a slow inco-ordination, the result of the irregular spasm, in which the over-action of the interossei is often conspicuous. There may also be inco-ordination, but rarely spontaneous movement, at the elbow and shoulder joints. The muscles may be of good size, and are sometimes even larger than on the healthy side, in consequence of the constant spasmodic exercise, and this even although the limb is shorter than the other. A slight tendency to similar over-action may sometimes be noted in the side of the face. It is very important to notice the side on which a slight movement is more marked. Such over-action often corresponds with the side chiefly convulsed, or on which an aura is felt, and is then of considerable significance as a sign of organic disease. In the foot the over-action is also often distinct, causing a tendency to inversion of the foot and over-extension of the toes.

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* 'Med.-Chir. Trans.,' 1879.

This mobile spasm is a frequent result of infantile hemiplegia, but its association with convulsions is not confined to cases of infantile onset. It was conspicuous in cases of convulsion in which the hemiplegia came on at seven, ten, eleven, twenty-one, and twenty-seven years, although there was no mobile spasm in the convulsive cases in which the hemiplegia came on after thirty.

The association of the two symptoms is too frequent to be accidental, and they are probably the result of the same morbid state. In most cases of mobile spasm after hemiplegia there is evidence that the lesion was vascular obstruction and not hæmorrhage or focal inflammation of the cortex. The damaged grey substance recovers imperfectly, and remains in a state of instability, over-acting spontaneously, or on the excitation of a volitional impulse. The conditions of onset of hemiplegia followed by convulsions always point to the same conclusion—that the initial lesion is cortical, and that the convulsions are the result of the imperfect recovery of damaged grey matter in the vicinity. It is of interest also to note that the common posture of the hand in a case of post-hemiplegic mobile spasm, pronation, strong flexion of the wrist, interosseal position of the fingers, is precisely that which is produced by the spasm of an ordinary epileptic fit.

In many of these organic cases, especially in early life, initial palsy is not recognised. It may readily be overlooked in an infant, especially when prostrate with some general illness. Moreover, a paralysing lesion destroying part of a motor centre is less likely to be followed by convulsion than one in the vicinity of the centre, which slightly damages its structure and deranges its functions. Initial paralysis is also more likely to be overlooked when this chiefly involves the leg, partly because less conspicuous, but especially because compensation by the opposite hemisphere so readily occurs in the case of damage to the leg centre, particularly in early life.

Sensation on the paralysed side is usually normal. In a few cases I have found a slight persistent defect of sensibility to touch. In no case was there any affection of the special senses. In rare cases, however, hysterical hemianæsthesia may occur in later life, and may demonstrate its nature by changing its side.

The convulsive attacks vary much in character. In a few cases the convulsion is general from the first, and similar to the attacks in idiopathic epilepsy. In the majority of cases the convulsion begins in, or is confined to, the paralysed side. But when the first fits are very severe they may be general in distribution, although afterwards unilateral and often beginning locally. In many, the convulsion affects all parts of the paralysed side, and does not spread to the other; in others, the unparalysed side is involved secondarily. In a few, only part of the paralysed side is involved, usually the arm, or arm and side of the face; less commonly the convulsion is confined to the leg.

A deliberate onset is present in a much larger proportion of cases of organic epilepsy than of the idiopathic form. The convulsion commences in one of the paralysed limbs in a considerable proportion of cases, and this deliberate commencement constituted the warning in most of the cases in which the patient was conscious of the onset. The commencement is most frequently in the upper limb, especially in the hand, often by a sensation which may pass up the arm and down the side. The early (recurrent) attacks may be so slight that their nature is not suspected, as in a patient in whom they consisted only of numbness of the thumb and forefinger, with brief powerlessness of the limb, for three years, and then convulsion occurred, spreading through the whole side. In some cases the commencement is by distinct spasm in the hand (twitching of the fingers, twitching of the thumb, and closure of the hand). Much less frequently the onset is referred to the arm as a whole; as by a sudden flexion of the arm.

Sometimes the commencing fit is first felt in the lower limb, and the initial symptoms are in the foot, usually a sensation in the toes, less commonly spasm. The sensation usually passes up the leg and side and down the arm, followed by spasm. In one case the warning was "pins and needles," felt simultaneously in both arm and leg. Commencement in the face is much less common. In one case it was twitching of the left side of the face with inability to speak. In another the initial symptom was rotation of the head.

In all the cases commencing in this manner the convulsion was confined to, or involved first, the paralysed side; in a large number the unparalysed side escaped altogether. In the case in which the fit began in the face, and in one in which it commenced in the hand, it did not extend beyond the arm.

Fits commencing in the hand or the foot appear, for some reason, to be much more frequent as a sequel of right than of left hemiplegia. On the other hand, in the cases in which the convulsion began in the upper part of the arm and in the face, the affection was almost as frequent on one side as on the other.

Ocular warnings are much less common in these cases than in idiopathic epilepsy. In one the initial visual sensation was "a fire before the eyes, going to the left," and the fit was left-sided. In one patient there was an ocular sensation, "as if the eyes were riveted together," followed by right-sided convulsion. A vague feeling of illness, vertigo, headache, vomiting, and a sensation of choking, are rarer warnings. An epigastric aura is rather more frequent, and may occur in cases in which the convulsion is unilateral. In one patient the attacks at first commenced deliberately in the hand, but afterwards by the epigastric-throat aura only.

The convulsion in these cases usually consists of both tonic and clonic spasm, and is of the same character as in ordinary epilepsy. Sometimes it is clonic only or begins by clonic spasm, afterwards becoming tonic. Consciousness is usually lost; sometimes, during a slight attack, it is unaffected.

After a fit is over, the state of the patient is similar to that after attacks of idiopathic epilepsy, but temporary weakness, "post-epileptic paralysis," is especially common on the side chiefly affected.

Minor attacks are frequent in cases of organic epilepsy, and usually consist in the local symptoms, spasm, or sensation, by which a severe fit commences, often without loss of consciousness. Sometimes, instead of any spasm, an initial sensation in the limbs is followed by transient powerlessness, apparently the result of inhibition of the motor centres. Not rarely attacks of *petit mal* occur which are quite similar to those of the idiopathic form—sudden or momentary unconsciousness, a sudden start, sometimes accompanied by an epigastric sensation. The convulsion tends to become, in the course of time, like that of idiopathic epilepsy, general, the brain being, as it were, trained to a more general discharge by the repetition of the process.

Hysteroid seizures are not uncommon in these patients. They may succeed epileptic attacks, major or minor, or may occur alone. Some of the most severe hysteroid fits I have seen were in a girl the subject of infantile hemiplegia, whose arm was permanently paralysed. The attacks come on without any indication of initial epileptoid seizure, and I have known them to continue, unless interfered with, for several hours—violent opisthotonos, bounding movements, biting, tearing of hair, &c. The same patient had also other hysterical manifestations, attacks of violent laryngeal spasm, rapid breathing, and a phantom tumour. But she had also severe epileptic fits beginning on the paralysed side.

In the cases in which the hemiplegia comes on early in life, mental defect is very common. The development of the whole brain seems to be retarded by the lesion, and the intellectual power is permanently below par. The patients are often mischievous and troublesome during childhood, and subsequently vicious, and often immoral.

CHAPTER VI.

HYSTEROID OR CO-ORDINATED CONVULSIONS.—HYSTERO-EPILEPSY.

GENERAL CHARACTERS OF ATTACKS.*

THE characteristic of the convulsions which are called "hysterical," "hysteroid,"† "hystero-epileptic," or "hysteria major," is, as stated in the introductory chapter, that the spasmodic movements are of a more or less co-ordinated character, such as may be produced by the will. Hence the spasm has what may be termed a "purposive aspect." Rigid fixation of the trunk and limbs alternates with wild movements, in which the limbs are thrown about; the arms strike out, the legs kick, the head is dashed from side to side. In the rigid spasm the back may be arched, even so that the patient rests on the head and the heels, or the trunk may be straight, and the limbs extended, less commonly flexed. The tonic spasm may be broken by quick quivering movement, a form of clonic spasm, but unlike the clonic spasm which is characteristic of epilepsy. Sometimes the spasm is rather of the character of rapid, rhythmical, quasi-volitional movements than of true clonic character. Laryngeal spasm, in some cases, forms a conspicuous feature of the attack. These phenomena are interrupted by periods of comparative tranquillity, attended in many patients by delirium, and sometimes by manifestations of emotion, usually of fear, which become more and more intense until they culminate in a paroxysm of convulsion.

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* The following description of the symptoms of these attacks is based on the careful simultaneous record of cases under my observation, some of which are described in detail. They will be found, I think, to give a faithful presentation of the chief features met with in this country, which are not quite the same as those that have been described in France. Several detailed descriptions given in the first edition of this work are here omitted, as being on the whole unnecessary.

† The term "hysteroid" was, as already mentioned, introduced by the late Sir W. Roberts as a designation for the more violent attacks, and is convenient as avoiding the trivial associations of the word "hysterical."

At the onset of one of these attacks there may or may not be a warning sensation. The patient often falls to the ground, but not with the sudden violence with which an epileptic falls. There is scarcely ever injury from the fall, which is often a sliding down rather than a fall. The violent co-ordinated movements may commence instantly, as soon as the body touches the ground, or there is an initial period of tonic rigidity, sometimes succeeded or accompanied by the quick clonic spasm above described, before the co-ordinated movements come on. It is important to note that such initial tonic spasm may, however, be the indication of a slight epileptic attack, to which the hysteroid convulsion is secondary.

Consciousness is rather changed or impaired than lost during these attacks. A sort of automatic consciousness, if the expression may be allowed, is present, but not that which is necessary for the subsequent recollection of the mental states which arise during the attack. After a fit is over, therefore, the patient may remember nothing about it. In many cases the patient seems unconscious during the paroxysms of violence, but in the intervals appears rational, although neither is afterwards remembered. During the intervals a peculiarity of speech and manner shows that the end has not yet come, and presently a fresh paroxysm occurs. The duration of the attacks varies from a few minutes to several hours, unless they are cut short by treatment. At the end the patient usually goes to sleep for a few minutes and wakes up well; or the attack may suddenly cease without sleep.

These attacks vary greatly in severity and character. When slight they are of the trifling character popularly known as a "fit of hysterics," into which an emotional patient will "work herself up," and in which there is no distinct affection of consciousness. When severe, the violence of the spasmodic movements is almost inconceivable. The head may be dashed against the floor with great force, and the body be alternately bent forwards and then thrown back in opisthotonos, so that the patient almost bounds off the bed. The more restraint is used, the more is needed, and these are the attacks in which the friends of a patient will tell us that "six persons had to sit on him to keep him down."

Similar variations are seen in the mental disturbance which attends the attack. This may be trifling, and amount only to an abnormal emotional state, or it may be so severe that for a time the patient is in a state of maniacal frenzy. Between these extremes every variety of mental disturbance may be met with. Some patients will scream loudly throughout an attack. Others will talk in an unnatural manner. One girl always counted backwards with extreme rapidity. In some patients a curious condition is observed, which may be termed therio-mimicry. The noises and actions of animals are strangely imitated. The patient mews like a cat, or, more commonly, barks like a dog. Even more frequent is a tendency to bite. Patients sometimes, but rarely, bite themselves. I have known the lip to be bitten, and two patients bit their own fingers, in one case so severely as to leave a permanent scar an inch long. Very commonly, however, the patients try to bite other persons, and the tendency renders considerable care necessary on the part of the attendants. Not only do they bite, but they do so in a curiously animal manner. A case is described in a subsequent page in which a lad of sixteen, after failing in an attempt to bite an attendant's hand, seized the corner of his pillow between his teeth, and throwing his head back, shook the pillow, just as a dog shakes a rat, or as the lion shook Livingstone. This therio-mimicry may be in part truly mimetic, but it seems to be, in part, the manifestation of some strange animal instinct which we possess in a latent or modified condition. A lad of twelve was found, in one attack, in the act of "worrying" a cat, having seized it by its neck with his teeth. The cat was making a vigorous defence with its claws, but was nearly killed before it could be extricated.

Such hysteroid convulsions may, and often do, occur alone the patient presenting, in the attacks, no other phenomena, and having no other kind of attack. But they also occur, with considerable frequency, in epileptic patients after genuine epileptic seizures. When a slight epileptic fit is over the patient passes into this condition of hysteroid convulsion, often with alternating delirium. The condition lasts for a variable time, a few minutes to an hour or more. These post-epileptic paroxysms seldom occur after severe attacks, but they are common after slight seizures. A patient may, for instance, have attacks of distinct epileptic character, slight or moderate in severity, preceded by an aura, and attended with loss of consciousness or with slight characteristic convulsion. Then, after such attacks have occurred alone for years, each of them, or some of them, are followed by this hysteroid convulsion. The epileptic attacks are of the same character as before, but after each, instead of simple sleep or quick recovery of consciousness, the patient presents the co-ordinated movements, throwing about of limbs, opisthotonos, and delirium, which have been described. Experience shows that in most cases in which such hysteroid attacks recur during many months or during years, they are not simple, but post-epileptic. At other times the patients have attacks of minor epilepsy without the hysteroid sequel.

In the pure hysteroid attacks which occur in the natives of this country, the initial tonic stage bears little resemblance to the spasms of a typical epileptic fit. There is not the irregular strained fixation of limbs, and deviation of head and eyes, which occur at the onset of the epileptic seizure, nor is there the violent shock-like clonic jerks, commencing gradually, and diminishing in frequency while they maintain their force. But in individuals of some other races, and especially in the French patients whose cases have been so carefully studied at the Salpêtrière, the disease attains a greater degree of intensity than in this country. In them, as is shown by the description of the disease given by Charcot, and especially by Richer in his recent admirable work on the disease,[*] the attacks commence by a convulsive seizure resembling a true epileptic fit somewhat closely The patient suddenly falls unconscious, in general, severe, tonic spasm, with deviation of the head, and the tonic spasm passes gradually into clonic spasm, succeeded for a few seconds by coma and stertor. This constitutes the first or "epileptic" stage. Then violent co-ordinated spasm comes on, the stage of "*grands mouvements*," opisthotonos, bounding movements, etc., such as have been described above, but of extreme violence. This is succeeded by a period of mental and emotional disturbance, with manifestations of joy, anger, or erotism—the stage of delirium. Sometimes there is a final stage of terrifying visions.

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[*] 'Études cliniques sur l'Hystéro-épilepsie,' Paris, 1881.

It may be asked, is not this merely the sequence already described—a true epileptic fit succeeded by post-epileptic hysteroid phenomena? Charcot and Richer have pointed out that, close as is the resemblance of the first stage to an epileptic fit, there are certain important distinctions which indicate an essential difference. (1) The attack is often preceded by a peculiar mental state, with hallucinations, and frequently accompanied by transient contractures of one or another limb. This condition gradually increases in intensity until the attack comes on. (2) The tonic spasm with which the epileptoid stage commences is usually immediately preceded by violent movements of the limbs. (3) An attack may be brought on by compressing the ovaries, or in some cases by touching certain "hysterogenic points" on the surface. (4) At any period of the attack, even of the epileptoid stage, it may instantly be arrested by ovarian compression. (5) These attacks are not influenced by such treatment as does good in epilepsy. If they are cured, it is by the treatment suitable for hysteria.

The crucial test of Charcot, compression of the ovaries, is rarely successful in this country. An attack can scarcely ever be thus induced, and although it may sometimes be arrested by this means, the effect is not sufficiently constant to possess any diagnostic value. It is certain that in England attacks presenting an actual combination of true epileptic and hysteroid symptoms are so extremely rare as scarcely to merit consideration. We have therefore no justification for the use of the word "hystero-epilepsy" in the sense it bears in France.

ILLUSTRATIONS OF HYSTEROID FITS.

Some of the characters of the attacks, of which a general description has been given, may now be considered in greater detail, and at the same time their features, as they occur in this country, may be illustrated by the narration of some cases.

Post-epileptic Hysteria.—Instances of the occurrence of hysteroid convulsion as a sequel to true epileptic fits may be first mentioned.

The first case is an example of such convulsion after more severe epileptic attacks than usually have this sequel. But it is instructive on account of the brevity of the hysteroid disturbance, which was, as it were, interposed between the epileptic fit and the subsequent sleep.

A pale, delicate-looking girl, aged 17, whose father had been insane, had suffered from fits since six years of age, at intervals of two to eight weeks. All were severe attacks; without warning, she fell with a scream, was convulsed, bit her tongue, and slept afterwards. About the age of puberty the fits, as they occurred before, became succeeded by hysteroid convulsion. In one which was seen, she suddenly fell with a scream, the head was turned to the left, the limbs rigid, the face became cyanotic, clonic spasm succeeded, she frothed at the mouth, and bit her tongue. The fit lasted exactly two minutes and a half. She then lay unconscious, breathing heavily, and still frothing at the mouth, for three minutes. Then she opened her eyes, looked strange, kicked and threw her arms about, and dashed her head down and arched her back for four minutes. She then seemed more conscious, but presently again went to sleep, and slept heavily for an hour, and then woke up with a severe headache.

A girl aged 14, without inherited tendency, became, at 13, subject to fits, evidently, from the description, severe epileptic fits, accompanied with tongue-biting. The severe attacks ceased, but attacks of *petit mal* continued, of which she knew nothing, and these, after a time, were succeeded by hysteroid convulsion. She would suddenly bend forwards, unconscious, be laid on the floor and lie still for a few seconds, without any rigidity; then become stiff and begin kicking with her feet and fighting with her hands for a few minutes, and then could be roused.

A girl aged 14, who formerly had severe epileptic fits attended by tongue-biting, came under treatment for seizures of the following character. While speaking to me she suddenly stopped and bent forwards, remaining still for a few moments without rigidity—a very common form of *petit* *mal.* Then she suddenly began fighting with her arms and stamping with her feet, and became stiff and rigid. The attack lasted only a few minutes, and she afterwards remembered nothing of what had happened.

Hysteroid convulsions often come on during sleep. In most of these cases there is reason to believe that the attack commences with an epileptoid seizure. It is, indeed, always difficult to exclude this, since the initial stage is scarcely ever seen, and when it can be observed, the conditions are seldom such as to secure accurate observation.

During the waking state some sensation may attend the onset. In many cases with a distinct aura, it is probable that the warning is really that of a true epileptic seizure, to which the hysteroid convulsion is a sequel. But a special sensation may occur before hysteroid attacks in which no epileptic element can be discerned. The most common warnings in such cases are a general feeling of malaise and illness, palpitation of the heart, giddiness, some cephalic sensation, an aura referred to both feet, and especially a sense of constriction in the throat, allied to or identical with the "globus hystericus" —a sensation which is not, however, confined to these cases.

Patients who are aware of the onset of an attack are often able to ward off its occurrence, sometimes altogether, sometimes for a time, by exertion, excitement, or by an effort of the will.

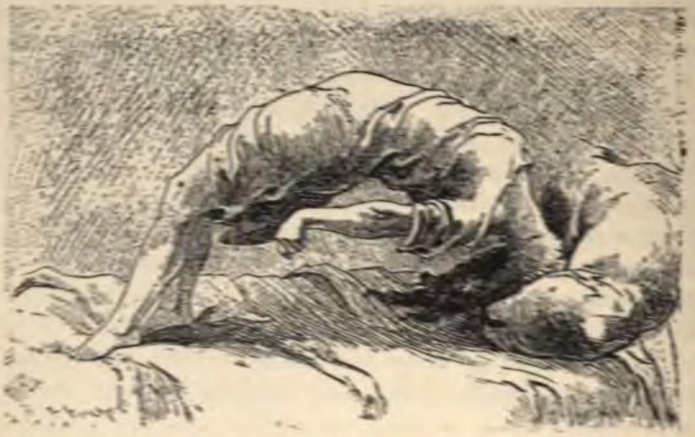
A young man aged 19, who had had many hysteroid attacks both day and night, preceded by giddiness, one day, when going home from the City to Norbiton, where he lived, felt suddenly giddy while walking to the station (at 5.30), and the giddiness continued for a quarter of an hour. He reached the station and got into the train, and then felt a "kind of stupor," which was so bad that at Clapham Junction he got out and sat there for an hour and a half. He then felt better, got into another train, and went home. As soon as he reached his house (at 7) he lay down on the bed, and the attack at once came on. He made a moaning noise, and began talking with great volubility about business matters; then he became violent and tried to hit his head, bit his lip, plunged and kicked for three hours and a half, and then went into a sound sleep.

It is probable that in this case the initial vertigo is evidence that the hysteroid convulsion was post-epileptic. Attacks of certainly epileptic nature may sometimes be postponed for a short time by an effort of the will aided by external circumstances. Moreover patients with such successive attacks are also liable to isolated hysteroid convulsions, especially when repetition has trained the brain for their development.

In most cases there is no change in the colour of the face when the attack comes on, but sometimes there is initial pallor. A fall is gentle usually. An initial scream, which may be repeated during the course of the fit, is an emotional cry, not like the strange laryngeal sound often heard at the onset of an epileptic fit. The rigid spasm may not commence until the patient has fallen, especially when the fall is gradual. In some cases the tonic spasm distinctly commences before the fall, which is, in these cases, more violent. When a patient falls violently, unconscious and pale, and remains for a few moments still, before the distinctive hysteroid symptoms come on, it is almost certain that the initial phenomena are those of an actual attack of epileptic *petit mal*, and that the hysteroid convulsion is thus post-epileptic. Such patients usually have other attacks without the hysteroid sequel.

Tonic Spasm.—When there is a stage of tonic spasm, the limbs are usually rigid in extension, the toes pointed downwards. The arms may lie by the side of the body, or at right angles to it, the patient, in the latter case, lying on the back in the attitude termed by Charcot "crucifiement." In some cases the arms may be flexed at the elbows. The fingers are usually flexed at all joints, the fists being clenched. The wrists are sometimes also flexed. There is never the "interosseal position," flexion at the metacarpo-phalangeal joints, which is so common in epileptic seizures. The duration of the simple tonic spasm is usually one or two minutes.

Opisthotonos.—The opisthotonic spasm is one of the most characteristic features of hysteroid convulsion. It is termed the "*arc en cercle*" by the French writers. It rarely occurs at the commencement of the fit. The initial tonic spasm may pass into it, or it may succeed the stage of clonic spasm, and often recurs from time to time during the progress of the fit. It may only be simple slight arching of the spine, as the patient lies on the back, or it may be such that the patient rests on the back of the head and the heels, and in extreme cases the trunk may then be pushed up by the feet, so that the neck is flexed to the extent that the vertex or even the forehead is the anterior point of support, and it seems almost as if the neck would be broken. Whilst it lasts the patient may suddenly sit up in bed, and then bound backwards into the rigid arch, and sometimes repeats the action with great force many times.



This representation of extreme opisthotonic spasm is one of Richer's admirable illustrations to his work on 'Hysteria.'

The opisthotonos may occur as the patient lies on the side. The knees are suddenly flexed, the spine bent, and neck bent backwards, in what Richer terms the *arc en cercle latéral*.

Many of the cases here narrated illustrate this form of spasm, since it is present in most severe hysteroid attacks. It was a very conspicuous feature in the following cases:

A girl aged 15 had had frequent attacks of the following characters. She suddenly screamed out and fell backwards, without pallor of the face. She lay quiet for a few seconds, apparently unconscious. Then she moved her hands like a child trying to box, gently at first, and then more violently, in "fighting" movements, at the same time kicking out with her legs. The neck and back were at first rigid and straight, but presently the [illegible] ceased and the back was arched, the patient resting on the head and [illegible] and from time to time the trunk was pushed up with the feet, so that the neck was bent backwards to such a degree that it seemed as if it would be broken. From time to time she would bound forwards, almost off the bed, and then back again in opisthotonos. After about five or ten minutes of this she suddenly became quiet, but talked strangely, and once alluded to the fit as occurring in another person, and said to the nurse, "Why don't you hold her down?" After a few minutes' quietude she again passed into a paroxysm of spasm, beginning with the shaking of the hands. This alternation recurred many times. In some paroxysms she tried to bite any one near her, or her pillow. From time to time there was strong internal strabismus, lasting a few moments, and then ceasing. The pupils varied, being sometimes widely dilated, sometimes extremely contracted. Such attacks often lasted for an hour or two. In some the paroxysms of spasm were much shorter than in others, lasting only half a minute. After an attack she had no recollection of it. This patient's attacks had always been of this character.

A girl aged 25 had frequent attacks during the six months preceding her admission. She had also persistent internal strabismus. The following is an example of the attacks. While lying in bed the internal strabismus became more intense, the angles of the mouth were drawn down. Then she suddenly struck out with her arms, while her face assumed an expression of alarm and determination. She became rigid, and turned over on to the left side, and her back and neck became strongly arched. The fingers were flexed, sometimes both wrists also flexed, at other times one wrist extended. This rigid spasm alternated with violent "fighting" movements. In some attacks the legs were throughout rigid and extended, in others they were, from time to time, kicked violently. The attacks lasted about ten minutes, and then ceased suddenly. There was no talking or biting or evidence of consciousness. Her sight was dim in the intervals (perhaps from spasm of accommodation), but this amblyopia ceased suddenly after one fit.

Clonic Spasm.—The movements in the clonic spasm are quick, small in range, maintaining the same frequency, and would often be more accurately termed tremor, or quivering, than clonic spasm, as in the following case:

A girl aged 18, without neurotic heredity, had suffered for eighteen months from fits of the following character. While sitting before me the fingers gradually became flexed, the elbows bent, and the hands were jerked very quickly. The legs were extended and rigid, the feet also extended, the toes pointed downwards. In a little time the legs were also jerked in fine, quick, tremor-like spasm. Then the spasm ceased; she sat up, looked wildly about, and presently passed into another paroxysm of rigidity and tremor. Strong ovarian compression lessened, but did not arrest the attack although it could be at once cut short by faradisation of the skin.

Sometimes the clonic spasm affects chiefly the orbicularis palpebrarum. It may amount to merely quivering of the eyelids, or to quick opening and shutting movements, occasionally combined with opening and shutting movements of the hands. Both of these are common and characteristic of the hysteroid state. They were well illustrated by the following cases. In the second, at least, it is difficult to exclude a slight initial attack of minor epilepsy.

A girl aged 15 had for twelve months one or two attacks a week, of the following character. She fell back rather gently, and then presented internal strabismus and rigidity of all the limbs in extension. From the first there was rapid opening and closing of the eyelids. Then the arms were thrown about, and she tried to bite those near her. Breathing was noisy, and the face became red. After a few minutes she became quiet and drowsy, but could be roused, and then passed into the same rigidity as at first, followed by similar movements, again to cease and to recur. Each attack lasted for an hour or more, in spite of douching and galvanism, until they were cut short by apomorphia in the method which is described in the chapter on "Treatment."

A girl aged 18, without inherited tendency, came under treatment for attacks which had commenced five months previously. Without warning she would fall back on the floor, being pale at first, and afterwards red. Then, lying on her back, the arms were stretched out at right angles to the body, in the attitude of *crucifiement*, the legs crossed at the ankles, the limbs being rigid, and the eyelids closed. On opening the lids, the eyes were directed straight forwards and the pupils were large, but instantly the eyeballs were turned upwards, strongly converging, and the pupils, with the convergence, became contracted. Then the legs were jerked with great rapidity, the feet being struck against the floor. The movement was small in range. Presently the right arm was moved in a similar manner, and then the left. Violent opisthotonos then occurred, the patient resting on the back of the head and the heels. After a few moments this ceased, the trunk falling suddenly to the floor, the quivering of the limbs returned, and the eyelids were similarly affected. These phenomena were repeated several times, the face being flushed. Strong ovarian pressure, at any time during an attack, caused the quick clonic spasm to cease, and induced straining of the muscles of respiration, and opisthotonos, which passed off as soon as the pressure was discontinued. No evidence of consciousness could be elicited. After these symptoms had continued for ten minutes, they ceased suddenly. The patient seemed conscious and sat up on the floor, but after a few minutes she fell back again in the same convulsive movements, but they were slighter, lasted a shorter time, and then ceased and did not again recur. She had such attacks every few days. The co-ordinated convulsions could be at any time instantly arrested by strong faradisation of the skin, the current being passed from the neck to one hand, and after a few attacks had been cut short in this manner they ceased to recur, and when she left the hospital she had not had an attack of any kind for three weeks.

In hysteroid attacks, although patients so often try to bite other people, and may even bite their own fingers, they do not bite the tongue. Tongue-biting is essentially an epileptic symptom.* But although the tongue is not, the lips are sometimes bitten. I have known instances in which patients, throughout every attack, persistently gnawed their lips.

The Co-ordinated Movements, which constitute so large a part of the phenomena of these attacks, are for the most part, as already stated, wild irregular movements, without definite sequence. Sometimes, however, some of the movements are repeated in rhythmical manner. It has been mentioned that the spasms in the arms or legs may have this character, sometimes quick, as in the case last described, sometimes slow and considerable in extent. The hands or feet may thus beat the floor. The rapid bounding movements of flexion and extension of the trunk, in which the patient alternately sits up and bounds back in opisthotonos (rarely seen in this country), are an extreme example of this form. More frequent are rhythmical lateral movements of the head from side to side.

Sometimes the movements consist of slow regular flexion and extension of the limbs. The legs may be thus alternately drawn up and pushed down, and the patient, if on the floor, may be propelled head foremost. Such slow movement of the legs may be associated with quicker flexion and extension in the arms. With similar movement in the arms, the legs may be rigid in extension. In one patient, after general tremor for fifteen or twenty seconds, the fingers were flexed, then the wrists, and then the elbows; then the arms, thus flexed, were drawn back at the shoulder-joint and thrown out in front, again to be flexed and drawn back and thrown forwards. This movement constituted all the observable motion in the fit.

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* Charcot states that the tongue may be bitten during the initial "epileptic" (pseudo-epileptic) stage of the severe attacks of hysteria major. In this country the statement certainly does not hold good.

A co-ordinated movement sometimes seen in these attacks is an attempt to "dig the hands" into the face or throat, which may result in serious scratches. It is common in post-epileptic states. For instance, a woman aged thirty-six, whose mother was epileptic, and mother's sister insane, had occasional fits since dentition convulsions. They usually occurred in sleep, but sometimes when awake, and then were preceded by a sensation of "air rising from the pit of the stomach to the head." In some of them she bit her tongue. In others, however, she merely fell and immediately began moaning, put both hands to her face and scratched it, and pressed her hands against her nose and mouth, as if trying to suffocate herself.

Laryngeal Spasm.—A somewhat rare variety of attack is characterised by extreme difficulty of breathing, not the rapid breathing sometimes observed in hysterical patients, but intense respiratory spasm. They are the "choking fits" of adults, and are termed "hysterical strangulation" by French writers. It is, no doubt, adductor laryngeal spasm, and may perhaps be an extreme degree of the functional disturbance which, in slighter measure, causes the globus hystericus. The patient with infantile hemiplegia and hysteria, mentioned on p. 163, had occasional severe attacks, very closely resembling prolonged paroxysms of laryngismus stridulus. One day she came into the out-patients' room in a state of hysterical rapid breathing—80 per minute. In order to ascertain whether it would persist during sleep, I hypnotised her by making her look at the hole in an ophthalmoscope. She was quickly asleep, and then breathed only twenty-five times per minute, but presently woke up in the most intense laryngeal spasm, with loud crowing inspiration, and congested and even cyanotic face. It lasted several minutes, and she occasionally hawked up bloody mucus. It would have been alarming had not its hysterical nature been recognised.

The following are other examples of this form of fit:

A girl aged 18. She was unconscious of any warning, but an observer could note a slightly frightened expression and restless movement of the eyes at the onset; then she would suddenly fall, and instantly begin labour-ing for breath. If placed on a chair, she could sit up, but the agony of dyspnœa was extreme. Her head was thrown back, her mouth half open, her arms held out before her with fingers separated; the muscles of inspiration were thrown into violent action, and a loud noise in the throat accompanied each laboured breath. From time to time there was an ineffectual attempt to swallow, and she would strike the upper part of the chest with violence, as if trying to shake something down. Her face rapidly became flushed, then dusky, and the dyspnœa increased, until she seemed on the point of suffocation. By closing the mouth and nose, and arresting respiration, the spasm could generally be stopped, but her aspect remained wild, she spoke strangely, and in a few minutes passed into another similar paroxysm. Even if placed under the influence of chloroform the dyspnœa returned as the effect of the anæsthetic passed away, and, if not stopped by means to be described, such attacks would go on for hours.

A girl, from 17 to 18 years of age, suffered from attacks which came on during sleep. Her mother, sleeping with her, would be waked up by a "suppressed choking scream," and find the girl unable to get her breath. She became dusky, then very pale, and then the spasm passed away.

It is instructive to compare the features of such an attack with the other well-known forms of respiratory spasm—the globus hystericus, the epigastric-throat aura of epilepsy, the paroxysms of laryngismus stridulus, and the respiratory spasm which constitutes so prominent a feature (with hysteroid convulsions) in the symptoms of hydrophobia.

Pharyngeal spasm also occasionally occurs, and may render deglutition impossible. It has been described, in France, as the "hydrophobic form of hysteria." The patients may be tormented with thirst, and yet unable to swallow a drop of water. I have only met with slight examples of this variety, but a graphic description has been published by Raynaud[*] of a case in which laryngeal and pharyngeal spasm co-existed with trismus, and the patient died in one of the terrible paroxysms of dyspnœa. The case is not altogether beyond criticism, but the balance of evidence is in favour of the accuracy of the diagnosis; the patient presented various hysterical manifestations, and a perfectly similar attack had occurred previously and passed away. The patient had, in the interval, become addicted to the hypodermic injection of morphia, and Raynaud suggests that it may be to the effect of this on the nerve-centres that the fatal termination of the attack was due.*

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[*] 'L'Union Médicale,' March 15th, 1881, p. 433. The discussion on the case at the Société Médicale des Hôpitaux will be found in the same number.

Eyes.—The eyelids, in hysteroid seizures, are sometimes separated, more often closed firmly. The eyeballs, as already stated, frequently converge strongly, as in several of the cases related, or are rolled up or to one side. The pupils vary; they never diverge; often they are large; sometimes they vary in size at different periods of the attack. They usually contract during the convergence of the eyeballs, and perhaps without convergence, from spasm of accommodation. The *reflex action* to light, when it can be tested, is usually preserved; but it is most difficult to make observations on this point, since the eyeballs are usually rolled up, and action to light cannot be satisfactorily tested unless the light falls on the macula lutea. Reflex action from the conjunctiva is usually lost or greatly diminished during the severer period of the attack. At other times it is normal. The loss seems to be part of the general diminished sensibility of the surface. In most hysteroid attacks a pin may be run into the skin without any expression of pain or reflex movement, and the conjunctiva may be touched without any contraction of the orbicularis. Sometimes there is a slight muscular contraction when the cornea is touched, but none when the finger is placed on the conjunctiva over the sclerotic, showing that sensibility, although lessened, is not abolished. So, too, although the skin may be insensitive to the prick of a pin, strong faradisation may be acutely felt. In the following case a careful observation on the pupils was made:

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* The opinion is supported by a remarkable case which has been under my notice. A woman, single, about 35, had become accustomed to morphia injections, and abductor laryngeal palsy occurred when she was not under the influence of morphia, ceasing entirely on its injection. During the intervals the characteristic inspiratory stridor was so loud as to be heard all over the house. The late Sir Morell Mackenzie examined the larynx during one of the attacks, and found almost absolute abductor palsy. Massage with isolation and hypodermic injections of strychnia effected a complete cure. Although no common symptoms of hysteria were present, the condition was clearly "functional," but not therefore devoid of danger. Death has occurred in apparently analogous states.

A girl aged 25 was admitted, after having been in almost continuous fits for a fortnight. She had no attacks for about three weeks after her admission, and then seizures occurred of the following character. She would fall gently, and lie flat on her back with the eyes open, the jaws separated and rigid, the arms by the side, and the arms and legs stiff. Then the arms, still rigid, were raised and dashed down, and the legs slowly drawn up and suddenly pushed down (as described on p. 175). The movements occurred at regular intervals of about half a minute. The attack was readily arrested by closing her mouth and nose. She had also attacks without this rhythmical movement, in which she fell, apparently unconscious, with body and limbs perfectly rigid, and mouth firmly closed. The eyelids were widely separated, and there was occasional twitching of the orbiculares palpebrarum and occipito-frontales. The eyeballs were slightly turned upwards. The pupils were equal, dilated, 6 mm. in diameter. They reacted well to light, although the conjunctiva was insensitive to touch. The respiration was quick and shallow. A little later the reflex action from the conjunctiva had returned. This attack was not cut short, and lasted three quarters of an hour, after which she slept soundly for half an hour.

Hysteroid attacks are, as several of the cases already mentioned show, common in young girls at ten or twelve years of age, and some of the cases which occur at this period are of great severity.*

Ovarian compression, which is so effective in inducing and in cutting short the attacks of hystero-epilepsy at the Salpêtrière, often, as already stated, fails to produce a marked effect in patients in this country, although ovarian tenderness is common. Sometimes, however, distress, choking sensations, and even an actual attack, may be produced by prolonged compression of the tender ovary, but its effect is not invariable. In a girl of sixteen, with well-marked hysteroid attacks, firm ovarian compression caused only pain in the head, faintness, and drooping of the eyelids. In another patient, aged twenty-five, in whom there was marked ovarian tenderness, pressure caused the breath to become short, the face to flush, and a peculiar feeling in the chest, such as often preceded an attack, but the compression never actually induced one. In another, however, during the paroxysms of wild movement, an attack of opisthotonic rigidity could always be induced by ovarian compression, but not at other times.

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* A remarkable and prolonged case of hysteroid attacks, of varied features, is described here at length in the first edition; but its interest, although great, is scarcely sufficient to warrant its repetition.

The striking difference between English patients and the neurotic French women, in whom a morbid train of symptoms has been elaborated by repeated induction, has often astonished French physicians who have visited this country.

Duration and Termination of Attacks.—The duration of hysteroid fits, when not arrested artificially, is very variable. They may last only a few minutes, but such brief duration is rare except in cases in which the hysteroid attack succeeds a true epileptic seizure. Unless cut short they often last ten, fifteen, or twenty minutes, and occasionally one or two hours.

They may be often arrested artificially by means which will be described more fully in the chapter on "Treatment," viz. closure of the mouth and nose, an abundant affusion of cold water, or painful impressions, of which faradisation of the skin is far more effectual, in this country, than ovarian compression. In some very rare cases these measures fail, and in such I have found the induction of nausea and vomiting, by a hypodermic injection of apomorphia, to be invariably successful.

The post-epileptic hysteroid convulsion can be arrested by these means as readily as the primary hysteroid attack. This sometimes leads to a mistake in diagnosis. It is said that a patient's fits were not epileptic because they could be stopped by water thrown on the face. An attack may thus be stopped, although the case is one of genuine epilepsy, the fact being that it is not the epileptic fit, but the post-epileptic hysteroid stage which is thus cut short.

The spontaneous termination of an attack is usually sudden; sometimes it is accompanied with a deep sigh, more often it is marked by a few minutes' sleep, at the end of which the patient sits up, and by the return of the natural aspect and tone of voice the friends know that there will be no recurrence. Occasionally the transition occurs suddenly without any other symptom.

Some patients seem to be aware of the termination of an attack; indeed, it occasionally seems as if the cessation was due to an effort of the will, under the influence of some obscure feeling that the attack is over. Of this the following case is an example:

A girl who had had a slight attack of hemiplegia at 3 years old, became liable to fits at 7, after a fall, and these continued until she came under treatment at 14. Whatever the earlier attacks had been, those which occurred under observation presented no epileptic character. They began with jerking of the head, and after this had lasted for an hour she fell down, and began kicking and struggling, throwing her head from side to side; occasionally she would take her pillow between her teeth and try to bite it, and try to tear her clothes. From time to time she rose up, and then dashed her head against the ground. Occasionally she laughed, and talked about a quarrel she had had at home. Suddenly she said, "Now I won't talk any more." She then slept for a few minutes and was all right, remembering nothing of the attack. She also had attacks of simple general rigidity (apparently hysteroid), and also some seizures in which there were quick flapping movements of the hands.

Death in Hysteroid Attacks.—As a rule, to which exceptions are infinitely rare, hysteroid attacks, however severe and alarming in aspect, are devoid of danger. The attacks of laryngeal spasm present the greatest apparent risk to life, and such paroxysms of dyspnœa as were presented by the hemiplegic girl mentioned on p. 163 are really alarming in aspect. That they are not absolutely devoid of peril is shown by Raynaud's case mentioned at p. 177; but this, I believe, is the only recorded instance of death in an attack of this description.

There is, however, another source of danger—the tendency to turn on the face which is sometimes seen in the post-epileptic state and has been already described. It is, strictly speaking, rather an automatic than a hysteroid phenomenon; but the two conditions merge into one another. An example of death from this cause has been already mentioned, and in the following case the same symptom was present. The patient was a little girl, aged twelve, who had had an attack of hysterical paraplegia, immediately cured by strong faradisation. Her fits always occurred on waking out of sleep, before she was quite awake. They commenced by a half moaning, half singing noise, and the louder this was, the worse was the subsequent attack. Then she gave a sudden spring and always turned on to her face, and presently began to scratch the pillow. Sometimes she would rise up in bed suddenly, and then dash her head back on to the pillow. The attacks lasted only a few minutes and always ended suddenly.

I believe that the attacks which occur during the transitional state, between sleeping and waking, are always really epileptic, and that obtrusive hysteroid symptoms are simply associated. In the following case death occurred in consequence of attacks which appeared, to those who witnessed them, to be hysteroid. The case was that of a little girl, aged eight, who had had attacks for three years, which began with a feeling of fright and giddiness, as if her head were being turned to the right—a characteristic epileptic onset. She always ran to any person near, and the convulsion never commenced until she got to him, however far she had to run. Then there was champing of the jaws, and the eyes were turned up so that the cornea was invisible, hands and elbows flexed, and the arms moved forwards and backwards at the shoulder for five or ten seconds. Such attacks sometimes occurred every five minutes. One day (at home), after a hearty breakfast, she had a slight fit, then a more severe one, and then, after some hours, another. All were of the same character as those in the hospital, but accompanied with profuse perspiration. She had had at home many similar series of long duration, some lasting nine hours. This series lasted for about four hours, and at the end of that time a rattle came in her throat, and she died almost immediately. No post-mortem examination could be held. The case was doubtless one of *status epilepticus*. It shows that repeated attacks which may be thought to be hysteroid should not be lightly regarded.

Temperature.—Hysteroid attacks, however severe and repeated, as Bourneville has shown, rarely raise the temperature, and never more than a degree or a fraction of a degree. In this respect they differ from true epileptic fits, several of which, occurring in rapid succession, may cause a considerable elevation of temperature.

In the patients who suffer from these convulsions, other symptoms of hysteria are frequent, and consist of the globus hystericus, aphonia, and the like, but these are usually slight in degree. Occasionally the patients present hysterical paralyses, contractures, or anæsthesia; but such symptoms are, on the whole, infrequent, and it is certainly more common, in this country, to meet with them apart from, than in association with, chronic convulsions. Of paralyses, paraplegia is perhaps the most frequent. Hemiplegia is rare. Spasmodic closure of the jaws is more common than contractures of the limbs; instances of both these, in males, will be described in the next section. Hæmianæsthesia is far less common than among the cases which occur in France.

HYSTEROID ATTACKS IN MALES.

Convulsive attacks of this class are sometimes met with in lads and young men. They may succeed epileptic seizures in the manner already described, or may seem to occur independently. The subjects of them may present other symptoms of hysteria, such as transient paralyses, or contractures of a limb, precisely similar to those met with in the female sex. The convulsive attacks present the characters already described, tonic and quick clonic spasm, opisthotonos, coordinated movements, and delirium with therio-mimicry. Their severity is often great; the frenzied violence of the movements and mental disturbance may be extreme, while the sex and strength of the patients render their management a matter of greater difficulty than in the case of females. The convulsions in the following case were among the most severe of the kind I have ever witnessed:

A young farmer, 15 years of age, six months before, had a "fit" in a dark cellar into which he had gone alone. Not returning, search was made, and he was found insensible, with a cut head. Whether he had fallen in a fit, or accidentally, could not be ascertained, but a few hours later a distinct fit occurred, and subsequently others. They were stopped by treatment, but recurred after a fright from a horse running away. They were often produced by excitement or by being startled, and he knew that an attack was coming on by palpitation of the heart. I was standing by him when one commenced. First one hand, and then the other, and then his trunk and legs became rigid. The mouth was twisted to one side. After a few seconds quick clonic spasm came on, commencing suddenly in all his limbs, and continuing with the same rapidity. In a few moments it ceased. Then he looked frightened, glanced around suspiciously, but tried to put out his tongue when told. Presently another attack of similar character came on, tonic followed by quick clonic spasm, and then a third, till he had had twelve. During the intervals there were manifestations of intense terror; the lad would rise up in bed, look fearfully to one side, catch hold of the attendant, whom he besought "not to let him be hurt." When the attack, or series of attacks, was over, he seemed to remember what had been said to him in the intervals, but did not recollect his own terror or the convulsive paroxysms. All his attacks presented the recurring tonic and clonic spasm, but the degree of maniacal disturbance in the intervals varied. In one attack, for instance, after the tonic and clonic convulsion was over—to quote my notes made at the time—"he lies quietly in bed for a moment, then suddenly fixes his eyes on some person near, or on the ceiling, or on some part of the room, and stares intently. A look of horror comes gradually over his face, becoming more and more intense as he slowly raises his head. Every muscle becomes fixed and rigid in an agony of terror; the sterno-mastoids stand out in the neck like bars of iron; his eyes seem as if starting from his head, his nostrils are dilated, and his breathing short and quick, while his heart beats with extreme rapidity. Suddenly he begins to struggle, to kick out with his legs, and to strike with his fists with tremendous force, from time to time throwing his arms and head back, still with the same expression of alarm. In a moment he is again rigid in tonic spasm, followed by the same quick clonic convulsion. When this is over, the face assumes a calmer aspect. Presently he starts up in the same paroxysm of terror, each inspiration now accompanied by a strange, weird cry, and his lower jaw drops from time to time, to be again jerked up. Suddenly he makes a gnash at the attendant's hand; failing to catch it he seizes the corner of the sheet between his teeth, and, throwing his head back, shakes it as a dog would shake a rat." This attack continued, presenting the same alternation of symptoms, for an hour and a half. On two occasions after an attack the jaws were closed in rigid spasm, so that it was impossible to separate the teeth; and this continued for three or four days until another attack, after which it had disappeared. On one occasion he had paralysis of the left arm for three days, which also passed off suddenly. The attacks ceased under treatment, but recurred for a short time, and then ceased permanently.

Attacks of a somewhat similar character occurred in a lad aged 15. Three months before coming under treatment (having been previously well) he had a prolonged encounter with some burglars, who broke into a house of which he, with some maid-servants, was left in charge. The lad had kept up for hours his endeavours to prevent the thieves from taking away their booty, and received several blows on the head, which stunned him for a time. He was found by the police next morning in some sort of fit; and fits recurred almost daily of the same character as those which he had after admission to the hospital, and of which the following is my note:—"He falls forward suddenly, turns over on his back, and immediately begins to struggle, throwing his arms about violently, and kicking. The eyes are sometimes open, sometimes shut. The eyeballs converge. From time to time the eyeballs are turned upwards or to one side, his face assumes an expression of terror, and he shouts out 'Murder! Robbers! Murder!' and then the struggling recommences. The rapidity and force of the movements of the arms and legs can hardly be described. There is no opisthotonos. His head is usually bent forwards, the jaw champed violently, and he is constantly trying to bite whatever is near him. Sometimes, after trying to bite, he makes a barking noise like a dog. After this has gone on for a quarter or half an hour, he suddenly curls himself up on his side, goes to sleep for a few minutes, and wakes up quite well, remembering nothing of the attack." In some attacks he made movements with the arms as if swimming, saying at the same time, "Swim away, swim away." The fits ceased entirely under treatment, and he remained well for four years, and then they recurred, similar in character but slighter. Treatment in hospital soon arrested them, and four years later he remained perfectly well.*

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* The following description of attacks, evidently hysteroid, is given in an account of a curious epidemic febrile disease, by Dr. Chas. Leigh, 'Philosophical Trans.,' No. 280, p. 1174 (Med. Essays, etc., abridged from the 'Phil. Trans.,' 1745, vol. i, p. 308):

"A poor boy of Lyme, in Cheshire, one John Pownel, about 13 years of age, was afflicted with the following symptoms:—Upon the crisis or turn of the fever he was seized with an aphonia, and was speechless six weeks, with the following convulsions. The distemper infested the nerves of both arms and legs, which produced the chorea, Sancti Viti, or St. Vitus's dance. The legs sometimes were so contracted that no person could reduce them to their natural condition. Besides these he had most terrible symptoms, which began in the following manner. He could perceive the fits to come on about the os sacrum or the extremity of the backbone, and the region of the navel, and then the disorder, as he imagined, united about the top of his head. He immediately afterwards fell into violent convulsions in the abdomen or lowest cavity, with that violence that sometimes two or three persons were forced to lie upon him to keep him in bed, his body being frequently raised from it. After this the nerves of the lungs were immediately affected, and then he barked in all the usual notes of a dog, sometimes snarling, barking, and at last howling like a hound. After this the nerves of the mandibles were convulsed, and then the jaws clashed together with that violence that several of his teeth were beaten out; and then at several times there came a great foam from his mouth. Afterwards he had an extreme wild look, snatching at anything near him, and would have tore off his flesh had he

The tendency to press the fists against the face, which has been mentioned as occurring in some hysteroid attacks in women, is also occasionally presented in males, as in the case of a young man who, at nineteen years of age, had had frequent fits for a year. "They begin with a feeling of faintness and heavy noisy breathing; he drops on his knees, clenches his hands, and drives his fists into his face. Then he makes a noise something like the bark of a dog, and begins struggling violently, shouts, kicks, and if placed on a bed rolls off it on to the floor, and, if left alone, would roll from one end of the room to the other, knocking his limbs violently against chairs and tables." The attack lasted only about a minute. Water at once arrested it.

The barking noise made by this patient, and which is not uncommon in hysteroid convulsions, is often associated with the tendency to bite, although the latter may occur alone. The barking and biting are prominent symptoms of the peculiar hysteroid state that occurs in true hydrophobia. A curious semblance of the genuine symptoms is sometimes presented by pure hysteroid attacks. In one patient, a lad of thirteen, attacks commenced after being bitten, and also frightened, by a dog. In the fits the boy would fall, throw his arms out, be still for a moment, and then pass into violent co-ordinated movements—kicking, bit-

not been prevented by the persons near him. This made me conjecture he might formerly have been bitten by a mad dog, which had introduced the hydrophobia; but I was convinced to the contrary, for I put a basin of water by him, and he was not in the least afraid of it, nor attempted to lap it. I saw him in three of these fits. But at other times in these convulsions he roared like a bull, made a noise like a hog, and sometimes like that of a gosling. . . . In a week's time I recovered the boy to his speech, his senses returned, his convulsions vanished, and the boy is now very cheerful."

An epidemic of hysteroid character is described in the 'Phil. Trans.,' No. 270, p. 799, "An Account of Uncommon Convulsions," by Dr. John Friend.

"At Blackthorn, in Oxfordshire, five little girls, 6—15, were seized with frequent fits of barking like dogs, together with violent motions of the head. . . . Dr. Friend visited another family at Blackthorn, where a boy and three girls had been seized with (similar) convulsions. . . . At first one of the girls was affected, and the first fit lasted for two hours; and the rest, as the mother informed him, were so struck with their sister's disorder, that in a few days they were also seized therewith" (Essays, etc., vol. i, p. 289).

ing, etc. He never bit his tongue. The initial symptom somewhat resembled epilepsy, but he never had any distinct epileptic seizures, and the attacks were not lessened by bromide. They ceased as soon as the boy was taken into a hospital, to recur when he returned home. After the attacks had lasted for several months, the boy's father, putting together the barking and biting, and the apparent cause of the first fit, came to the conclusion that his son was suffering from hydrophobia. Having applied without success to several hospitals, he went to a police magistrate and complained that he "had a boy who had been suffering for some months from hydrophobia, and no hospital would take him in!" It was in this lad that the animal tendency to bite was carried to such an extreme that he was once found in the act of "worrying" a cat.

In the majority of cases of such hysteroid fits in males, they are certainly post-epileptic, although the primary element may not be easy to discern. In a few they seem independent, chiefly in lads or young men of feminine mental characteristics, or in those who have been addicted to masturbation.*

MENTAL DISTURBANCE IN CONNECTION WITH HYSTEROID ATTACKS.

The mental disturbance which attends hysteroid convulsions is a very characteristic feature of the more severe varieties. It is seen in a very striking form in the French hystero-epileptics, whose convulsions are preceded and followed by hallucinations and emotional delusions, of which graphic descriptions and illustrations are given by Richer. But it is also, in a somewhat different form, a prominent characteristic of many hysteroid fits met with in this country, as several of the cases described in the preceding pages testify. During a series of fits, in the intervals between the convulsive paroxysms, there is often some mental derangement, and often positive delirium. The patients talk in an unnatural manner, and may have distinct hallucinations. Events which happen before them are falsely interpreted, and incidents lately past, which have made a strong impression on the mind, are reproduced and dominate their ideas. Their *bizarre* fancies are often more like the inconsistencies of a dream than like the delusions of the insane. In some cases the therio-mimicry is apparently the expression of strange hallucinations, to which the tendency to bite may be in part related. In most cases the disturbance of emotion is very marked. The ecstatic states common in these cases in France are very rare in this country, and erotic manifestations are practically unknown here. The emotion is usually painful—fear, horror, anger; and the convulsive paroxysms appear to be, in some cases, the effect of the culmination of this emotional disturbance.

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* The details of other cases were given in the first edition of this book, but as they illustrate no new features they are here omitted.

A close relation exists between these hysteroid states and epileptic mania. The delirium, which is so characteristic a feature of the attacks, often amounts to actual frenzy. The manifestations of emotion are more intense, and also more chaotic, than in ordinary delirium. The patient's condition might be described as one of compressed mania, the outbursts of varied feeling and action being too concentrated to permit of deliberate expression.

When a series of convulsions is over, the patient's mind usually returns to its normal state, but sometimes mental disturbance persists, in slighter degree, for a considerable time, and it may even come on apart from the attacks. In one child, with delusions after the fits, there were occasional periods of spiteful mischievousness or of a semi-demented state. I have known such patients to have transient periods of suicidal tendency, and even to attempt suicide, with the apparent intention of effecting it. In one case, a boy aged twelve years had frequent attacks, apparently hysteroid, before and after which he had attempted suicide, having tried to hang himself and to cut his throat. The first fit occurred six months before he came under treatment, after a severe fright. They began with an aura resembling that of epilepsy, a sensation in the left great toe which passed up the body to the head, when he lost consciousness and began "struggling." After admission into the hospital, however, he had no other fit, iron only being given.

How profound may be the mental disturbance in some of these patients is shown by the following case, in which the mental disturbance of hysteria passed into a condition of actual insanity:

A girl, aged 26, had suffered from fits, evidently, from the description, hysteroid, for six months. After admission she had several attacks in which she slowly fell backwards, without pallor, and then struggled and threw herself about, being made much more violent by any attempt to restrain her. Water quickly brought her round, and so did strong faradisation. In some attacks she made blowing noises with her mouth, and tore her hair. After a time the attacks were attended by more mental disturbance, and subsequently paroxysms of mental disturbance came on without any preceding fit. For instance, one day she had a fit, went to sleep for a short time, and then seemed well; she got up and dressed herself, but two hours later suddenly began to undress herself, and took off nearly all her clothes. She was prevailed upon to get into bed, but presently got out again, and began to take down the pictures in the ward, and if any person touched her she attempted to bite, kick, and scratch him. In the course of a few days she presented muscular weakness, which increased until she was in a most pitiable condition, scarcely able to walk, except during paroxysms of violence. On one occasion she had managed to slip out of the ward unobserved, and was found lying naked on the stairs. Under treatment she improved greatly, became strong, and passed a month without an attack, but afterwards had an outburst of such violence that it was necessary to transfer her to Bethlem.

After her admission there she remained without an attack, being quiet and industrious for seven weeks, when she suddenly became statue-like, standing fixed and taking no notice of her surroundings. After an hour of this condition she suddenly sprang upon the attendant, got her down, and would have injured her if not prevented. She then passed into a state of hysteroid convulsion, throwing the limbs about wildly, and clutching any persons near her, and trying to bite them. She was put into a wet pack. After a period of quiet she had another convulsive attack, and then remained still, except for singing hymns. The next day she was better, but other attacks recurred. Ultimately she had several recurrences of grave mental derangement, with normal intervals, and at last recovered, apparently, mental stability.

The case illustrates the close connection between the transient maniacal disturbance of hysteria and definite mental derangement. When such symptoms exceed in duration that which can be regarded as "paroxysmal," there is always danger of their persistence. It is most important to beware of ascribing mental symptoms to hysteria because they are accompanied by hysterical manifestations. These do not lessen the significance of disturbance which transcends the range of hysteria.

ATTACKS INTERMEDIATE BETWEEN HYSTERIA AND EPILEPSY.

Many careful observers, as for instance Trousseau, have considered that the characters of some attacks are intermediate between hysteroid and epileptic fits. But the tendency of recent study of these diseases has been to discountenance the idea that there are such attacks, to regard all doubtful cases as belonging entirely to one disease or to the other, or as presenting a serial (and not simultaneous) combination of the two. There is much, however, in the results of extensive observation which suggests the occurrence of such intermediate seizures. A comparison of the features of attacks which we consider epileptic or hysteroid, shows that some deviate very considerably from that which we regard as a typical seizure of either kind. In some epileptic fits, as part of the attack itself and not a sequel, there are phenomena which remind us of hysteroid seizures. I may refer, for instance, to the severe attack described on p. 96. It occurred in an hysterical girl, who had also purely hysteroid attacks, and others similar to that described. The onset was a sudden violent fall, without any premonitory symptoms; the clonic spasm was shock-like, perfectly epileptic in type, and the aspect of the patient was precisely like that seen in a most severe epileptic fit. And yet there was throughout a tendency to opisthotonos, and very brief hysteroid spasm came on without any interval, as the terminal stage.

Co-ordination of the muscular contractions which constitute the convulsion is the characteristic feature in hysteroid attacks; the absence of co-ordination is the characteristic of epileptic fits. But the initial phenomena of some epileptic fits—such, for instance, as the visual and auditory warnings previously described—show that the "discharge" in such attacks, when slowly developed and in certain situations, may be in the form of a co-ordinated nervous process. The same conclusion is suggested by some other initial symptoms, such as the act of running. An equally complex act preceded the attacks in a patient who had had major and minor attacks since rickets in infancy, and presented a family history of epilepsy. The minor attacks consisted of slight clonic spasm; the major were severe epileptic convulsions attended with tongue-biting. After both he went to sleep, but after the minor attacks, before going to sleep, he always jumped for five minutes, got on a table or chair and jumped off, and tried to jump over the banisters. *Both severe and slight fits began with hopping round the room on one leg.*

If the discharge may cause, in its commencement, a co-ordinated movement, it is not surprising that cases should be occasionally met with in which all the spasm of a slight fit of genuine epilepsy should consist of co-ordinated movements, *i. e.* that spasm of hysteroid type should constitute the convulsion of a true epileptic fit. I believe that this was the case in the patient whose attacks are described on p. 109. The fits occurred almost daily during several years. Sometimes they were excited by rising from the sitting posture, at other times they occurred apart from movement, both by day and night. There was no warning; the attack was very brief, lasting only a few seconds; the patient was unconscious, the pupils dilated, the conjunctiva insensitive, and after the fit there was almost complete loss of the knee-jerk for a few seconds. But the tonic muscular spasm, always the same, consisted in flexion of the left leg and elevation of the right arm; the right hand made scratching movements, so as sometimes to inflict a number of scratches on the face; the expression throughout was one of extreme alarm, and sometimes the tongue was protruded. Throughout two years the attacks varied very little in character. The character of the spasm, the emotional expression, the occasional protrusion of the tongue, were hysteroid, but the general course of the disease, the sudden brief fits, with complete unconsciousness and dilated pupils, rendered it much more closely allied to epilepsy than to hysteria. Of the latter condition there was no other manifestation. Moreover, the attacks ceased under bromide, and were entirely uninfluenced by any treatment for hysteria. The fatal case described on p. 182 was probably also an example of this intermediate form.

Emprosthotonic spasm, a tendency to "curl up in a ball," is a form of co-ordinated spasm occasionally seen in cases which seem to be of this intermediate class. For instance, a boy, aged thirteen, had suffered from fits for fifteen months, which commenced with a sudden start of considerable violence, and immediately his legs became strongly flexed, and his trunk bent forwards with the head between the knees. These attacks occurred chiefly in sleep, but he had somewhat similar attacks in the daytime, which commenced by a tingling sensation in the right temple; then he clasped his hands behind his head and bent his head forwards to his knees, kicking with his legs the while. He always passed urine during the fit. He also had attacks of sudden faintness during the day, but there was no trace of *petit mal* at the commencement of the co-ordinated fits, and it was during the progress of these that the micturition occurred. He had twelve or fourteen fits a day on various treatment. It was then found that he practised masturbation; a blister on the prepuce reduced the fits to from two to seven daily. He was then circumcised, and the attacks ceased at once, and did not recur.

These facts suggest, I think, that the morbid action of the nervous system which causes the visible phenomena of attacks may in some cases present such a combination of the processes which underlie the hysteroid and epileptic forms of convulsion, that attacks occur in which the characters of the two forms are combined at the same time, and are not merely associated in consecutive development. No doubt most of these cases may be placed, approximately, in one or the other group; but they show that the two forms of disease are not separated by any fixed and impassable symptomatic boundary.

CHAPTER VII.

CERTAIN MORBID ASSOCIATIONS OF EPILEPSY.

THE morbid states with which epilepsy may be causally associated have been for the most part enumerated in the chapter on "Etiology." Some others, however, deserve mention, the relation of which to epilepsy is uncertain in nature.

HEART DISEASE.

An abnormal condition of the heart is met with in many cases of epilepsy, but not in sufficient number to indicate a definite relation to the idiopathic disease. In some cases an imperfect supply of blood to the brain may have assisted in the degradation of nutrition and function, but is probably effective only in the presence of a congenital tendency, or of a powerful acquired disposition.

Organic heart disease does not seem more frequent in the subjects of epilepsy than would be expected from its general distribution. The cases are, of course, excluded in which recurring convulsions are the result of embolic hemiplegia.

The occasional occurrence of cardiac symptoms as the aura of epileptic attacks, gives some significance to a more definite relation between paroxysmal cardiac disturbance and convulsive attacks, such as the following case presented:

F—, aged 28. No neurotic family history or evidence of syphilis. She had had four attacks of rheumatic fever, the last nine months before. Soon after this attack she became liable to severe paroxysms of palpitation, chest pain, and dyspnœa. These continued for about two months, and then ceased on the occurrence of some convulsive seizures, which recurred almost daily up to the time of her admission. She was found to have a presystolic mitral murmur, not always audible. Occasionally a systolic murmur could be heard. The heart's action was frequent, 120—160; the pulse small and rather hard. There was no goitre. The convulsive attacks were said to be sometimes preceded by "swimming in the head," sometimes by no warning. In some of them she was said to have bitten her tongue. The first thing noticed was tremor in the right arm and leg, without change in the colour of the face. After a few minutes the head and eyes were turned to the left, the head was thrown back, the back somewhat arched, and the arms became rigid. Then hysteroid symptoms occurred, the arms were thrown about, she knocked her head, and pulled her hair.

The hysteroid symptoms of these attacks were cut short by stopping the breath and by water, and ceased. The pulse, however, continued to rise in frequency, being rarely below 130, and the attacks of dyspnœa and palpitation, which had occurred before the fits commenced, again returned. Many of these were carefully watched. She would suddenly press her hand over the lower part of the sternum, presently almost stop breathing, with teeth tightly clenched, and an expression of extreme alarm and distress on her face, rocking to and fro, and able to speak only a word or two in a whisper. She described a pain, usually behind the sternum, beginning gradually, and after a few seconds becoming most intense, shooting up to the left shoulder, and sometimes down the left arm to the tips of the fingers. Before she was conscious of the impending attack its approach could be recognised by a change in the character of her pulse. Previously 90—120, moderately soft, it would gradually become smaller and harder and more frequent, and then the pain came on. Towards the height of the seizure the pulse was 180—200, and extremely small and hard, but just before the attack began to subside the pulse could be noticed to become softer. Nitrite of amyl relieved an attack when flushing was produced, but it was difficult to get this result; bystanders several feet away were flushed long before the patient to whose nostrils and mouth the amyl was applied. The attacks continued in spite of treatment.

CHOREA.

Epilepsy and chorea are occasionally associated, and were so in twenty cases of this series. The relation between the two diseases was as follows. In eight cases epilepsy existed before the chorea. In twelve the chorea occurred first: in four of these the fits began at the time of the chorea, and persisted afterwards. In only two of these was there heart disease. In five cases the fits occurred long after the chorea, the interval being four, six, twenty-seven, thirty, and forty years respectively. It seems probable in the cases in which the fits immediately succeeded the chorea, and possible in the cases in which a short interval elapsed, that impaired nutrition of the motor centres during the chorea may have left a predisposition to further disturbance under the action of some other exciting cause.

The possibility of a close connection between the two forms of spasmodic disorder is shown in the following remarkable case, in which paroxysmal convulsive seizures were part of a severe attack of chorea. In them, although the spasm was in part that of chorea, so intense as to assume a convulsive character, there was also loss of consciousness and spasm of a tonic character preceding and accompanying the choreoid convulsion. The significance of this case is that the convulsion must be ascribed to the same morbid process as that which was the cause of the chorea.

Alice P—, aged 15, was admitted in October, 1875, with general chorea, very violent in character. She had had repeated attacks of chorea during the preceding seven years, for which she had been in several hospitals. She had a loud mitral presystolic murmur. At first the choreic movements were greater on the left side than on the right. The attack lasted four months, and then the chorea gradually lessened, almost but not quite ceasing for several months, and then increased so that in October, 1876, she was readmitted. There was then much mental obtuseness and a dull aspect. When she was at rest in bed the spasm was slight; on movement it became extreme and general. The speech was difficult. The tongue was protruded and withdrawn slowly. Almost every day after her admission she had a peculiar convulsive seizure. She gave a groan, became flushed and somewhat cyanotic; her head was turned to the right, the left sterno-mastoid being strongly contracted, and the eyes deviated to the right. There was a good deal of general movement of the limbs, at first choreic rather than convulsive. She was apparently unconscious. The movements increased, and she had some degree of opisthotonos for a short time, and the character of the movements became more convulsive. The condition lasted three or four minutes and gradually went off, leaving her in a dull, heavy state, and with more choreic movement than before the fit.

In another fit, which was watched from the commencement, the head again turned to the right. There was no initial pallor of face, but the countenance became more suffused than before the fit. The right arm and leg were affected with greatly increased choreic movements, while the left arm was stretched out rigidly from the side with the fingers extended, the thumb extended and drawn under the fingers. The rigidity was cataleptic in character, for the arm remained in whatever posture it was given. The fit lasted about two minutes, and left the patient very stupid and dull. After she came to herself the left arm was weaker than before.

A third fit which was observed differed in some respects. She seemed unconscious for half a minute, and then convulsion set in. The head was drawn violently to the right; the eyes remained open, the pupils rather dilated. There was some arching of the back, and tonic spasm in both arms and hands, that in the flexors preponderating, so that the elbow, wrist, and finger-joints were all strongly flexed, and the arm was drawn up almost to a level with the shoulders. After a few seconds the right arm became affected with a sort of choreic movement, the wrist and fingers remaining flexed. The left arm became more flaccid, but remained motionless, while the right arm was affected with this movement. The legs were not specially noticed, but there was certainly not much movement in them. The jaws all the time were firmly closed. The whole condition lasted about two minutes, and she was left dull and stupid.

Fits similar to these occurred daily for three weeks. Under hypodermic injection of morphia the fits ceased, and the left arm became stronger, but the choreic movements in it were more considerable. She improved for a time, and then relapsed and remained stationary until glycerine was given (half an ounce three times daily), which was followed by a very rapid and immediate improvement up to a certain point. Afterwards, with country air and zinc, all choreic movements ceased, the mitral murmur persisting. A year later, however, she was under the care of a colleague with another attack.

In the next case, also, the convulsive attacks, distinctly epileptiform, must be ascribed to the morbid process which caused the chorea, since they immediately succeeded the chorea and corresponded to it in distribution.

George C—, aged 12, presented himself with a history of a first attack of chorea, which had commenced six weeks before, and had lasted a month, leaving, however, considerable weakness of the legs, that of the right being the greater, and some weakness of the right arm. During the fortnight after the cessation of the chorea he had had several right-sided convulsive attacks, with loss of consciousness. Under treatment the fits became much less frequent, but did not cease; the muscular weakness, however, disappeared. The patient had no cardiac murmur, but the action of the heart was irregular.

The attacks which succeed chorea are, however, much less frequently epileptic than hysteroid, or of an intermediate form not quite corresponding to either. In the next case, for instance, the first attacks were apparently distinctly epileptic, but subsequent seizures, from their character and long duration, were unlike any ordinary epileptoid seizures.

The patient was a married woman, thirty-two years of age. As far as could be ascertained, there was no family predisposition to nervous disease. She had had good health in early life. At nineteen years of age she had an attack of chorea, occasioned by a fright, and sufficiently severe to cause her admission into a general hospital. The attack was a long one, lasting two years. Two years after the cessation of the chorea, and after some trouble, she had her first convulsive seizure. No fuller account of it could be obtained than that she was stiff, and screamed, and bit her tongue. The fits continued from that time; and she said that of late, when a fit was coming on, objects looked strange to her and she saw sparks before her eyes, and sometimes an appearance as of smoke or of "all kinds of colours." She was admitted into the hospital, and there had attacks in which, with apparent loss of consciousness, there was general tonic rigidity, but no clonic convulsion. Some of the attacks lasted several hours; others were shorter in duration, and seemed to be excited by a severe cough.

Another example of the association was presented by a patient whose case has been published elsewhere.* Epileptoid and hysteroid attacks occurred, but the patient suffered also from organic disease of the heart. The attacks in the next case were probably severe hysteroid seizures after an initial epileptic stage.

Ada S—, when five years of age, was under my care for a severe attack of chorea, from which she recovered, apparently completely. Three years after the chorea, when nine years of age, she became subject to "fits." These attacks commenced with jerking of the head and loss of power of speech for several hours. She was able during this time to hear quite well, but was unable to speak. Then she would suddenly lose consciousness, the jaws "champed," a noise was made in the throat, and "foam" collected in the mouth, almost choking her. There was no general convulsion. After the attack she generally slept for some time. At one time these attacks recurred regularly every three weeks. Under treatment the fits became less frequent and slighter, but there was much jerking of the head after waking in the morning, lasting an hour or more. In the slighter fits she would suddenly fall, be unable to speak for a moment, and rigid; then she would scream, clutch with her hands, and foam at the mouth. There was no cardiac murmur.

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* "On the Clinical History of Chorea," 'Brit. Med. Journ.,' March 30th, 1878.

In the following cases the chorea succeeded epileptiform attacks, and it is probable that the two were due to a common condition of cerebral instability rather than that there was any causal relation between them.

A single epileptic fit succeeded a month afterwards by hemichorea.—A lad aged 17 lately came under treatment for slight but characteristic hemichorea. The affection was of two months' duration, and was first discovered by objects being dropped in consequence of a "kind of catch in the hand." There was only a slight occasional twitch in the hand and on the right side of the face, but an attempt at movement was accompanied by characteristic choreic inco-ordination. The right hand was weaker than the left. (Dynamometer—right 31, left 41 *kilogrammes.*) He had never had rheumatic fever, and there was no cardiac murmur. His mother said that about three or four years ago he had an illness, accompanied by twitchings, and occasioned by fright. Three months before the patient came under observation, and one month before the commencement of the hemichorea, he had an epileptic fit, which seemed, as far as could be ascertained, to have been general. He fell without warning, and during the fit he bit his tongue.

Left-sided fits for six months, beginning in foot; slight attack of general chorea.—Eliza T—, aged 22, had her first fit six months before she came under treatment. The catamenia had never appeared. She had had no food for two days before the first fit. It was the same in character as subsequent attacks. Each began with a sensation in the left foot; the left leg was then drawn up, the knee being flexed, with a series of jerks, and the foot drawn up over the other leg. The sensation then seemed to pass up her side to the top of the head, and then back again to her heart, where it caused a sort of pain, with which she lost consciousness and fell backwards. What happened during the further progress of the fit could not be ascertained; apparently some time elapsed before consciousness was recovered, and on coming to herself she invariably asked the time. Two or three such fits had occurred weekly. For a few weeks before admission she had noticed a numb feeling in the left arm, and when holding an object the grasp would suddenly relax for a moment, and during about two weeks there had been involuntary movements in the arms. On admission there were well-marked choreic movements of the hands and head, slight but quite characteristic, spontaneous. There was very little inco-ordination on voluntary movement. There was no cardiac murmur. After her admission the chorea gradually ceased in the course of six weeks, and she had no fits, although she sometimes had a sensation such as preceded a fit, commencing in the foot and passing up to the head. These sensations continued for some weeks after her discharge; but she had no severe fit during three months in which she continued to attend as an out-patient.

Fits from infancy, chiefly left-sided; chorea at eleven, chiefly affecting left side.—A girl aged 16 came under treatment for epilepsy, which had existed from infancy. The first attack occurred at six months of age, during sleep. No history of rickets could be elicited. The attacks recurred at first every three months, afterwards more frequently. When eleven years of age she had an attack of chorea, of three months' duration, succeeding an illness with some symptoms of rheumatic fever. The chorea began in the left side, and afterwards spread to the right, but throughout it was the more severe on the left side. During the chorea she had no fits, but they recommenced immediately after the cessation of the chorea. The fits were preceded by no warning; consciousness was lost. The left hand was clenched and put out, the right being kept still. The mouth was drawn towards the left. There was usually little or no jerking, but sometimes there was a good deal, and it was then confined to the left side. There was no evidence of heart disease.*

MYOCLONUS MULTIPLEX.

The peculiar partial muscular contractions, widely distributed, and generally more or less symmetrical, which have been called "paramyoclonus (or myoclonus) multiplex," have been occasionally observed in association with epileptic fits.† Such cases have generally presented evidence of more considerable change in the cortex of the brain than is present in ordinary epilepsy. The association is apparently due to associated slight changes in the cerebral cortex and in the spinal cord.*

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* A case which, notwithstanding its complexity, illustrates the relation between convulsions and chorea, is that of a girl who had slight chorea at fourteen, and afterwards, at puberty, some transient attacks of mental derangement. At twenty-two she lost much blood at confinement; eight days later puerperal mania set in, followed, ten days later, by epileptic attacks. The urine was normal before and after delivery. She had 100 fits in eight days. Maniacal symptoms followed, and six days after the last fit chorea returned, and lasted for a few weeks (Easterbrook, 'Journal of Mental Science,' January, 1900). In a case of chronic chorea, associated with epilepsy, each epileptic fit increased the severity of the choreic movements (Bechterew, 'N. Cbl.,' 1164).

† Cases recorded by Unverricht ('Die Myoclonie,' 1891), Brisoles ('Neur. Cent.,' 1896, p. 1015), and four cases in one family mentioned by Mott (Croonian Lectures, Royal College of Physicians, London, 1900, Lecture IV). In some of these cases the character and energy of the muscular contractions resembled those of "electrical chorea" rather than typical "myoclonus."

PAROXYSMAL SNEEZING.

Among other paroxysmal disturbances in the subjects of epilepsy is paroxysmal sneezing. It is a rare association. A girl with infrequent but well-marked epileptic fits constantly sneezed violently six or eight times after waking in the morning, and sometimes woke up in the night with a paroxysm of sneezing. There was no local nasal symptom. In another patient a violent sneeze often occurred at the onset of a fit, and sometimes a similar sneeze was followed by a moment's unconsciousness.

ENLARGEMENT OF THE THYROID.

The thyroid is occasionally enlarged in epileptic females who are in the first half of life. It is usually slight in degree, and unaccompanied by cardiac or ocular symptoms, but may be increased after a fit, and especially after a series of attacks at short intervals. Occasionally the enlargement is associated with frequency of the heart's action, 120—160, with general arterial pulsation, and sometimes with distinct prominence of the eyes. In one case, a young girl with epileptic fits from eight months, all these symptoms were conspicuous, but were stationary in degree, save for transient increase after a fit. The child's mother had suffered from characteristic exophthalmic goitre from the age of nineteen to twenty-four, and had then recovered perfectly. I have never met with progressive symptoms in cases of this association, but some enlargement of the thyroid will be found, if looked for, much more frequently than might be thought, in the subjects of epilepsy. I have met with a few instances of such enlargement in cases of the pigmentation of the skin which is so often caused by arsenic given regularly to prevent bromide rash, but in two these were probably only coincident.

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* The inference has been drawn from the association that the epilepsy, in such cases, is of spinal origin. Further, however, there seems no justification.

MIGRAINE.

Migraine is often related to epilepsy. We cannot practically separate the paroxysmal headaches that are unilateral, and literally "hemicrania," from those which are general, since both may be preceded by visual or other sensory prodromata, and may entail the vomiting which has obtained for the attacks the designation "sick headache."

The common relation is one of substitution. A patient suffers from such periodical headaches, often from early life, and these cease on the development of epilepsy. When they do not cease, they become much less frequent. My notes contain many such cases. A girl suffered from occasional severe headaches, preceded, for five minutes, by bright lights before the eyes, until epilepsy set in at seventeen, when the headaches entirely ceased. Another patient became subject, at fifteen, to frequent severe, unilateral headaches, preceded by hemianopia. At twenty-two, epileptic attacks began and the headaches became very rare. The same relation is seen with pain of more neuralgic character. In one case paroxysmal attacks of severe pain over the eyebrows and across the bridge of the nose occurred twice a week until epilepsy supervened, and then ceased entirely. In another, paroxysmal headaches, preceded by dimness of sight, from early life, ceased when epileptic fits commenced at thirty, but returned at thirty-two on the arrest of these by treatment. So, in a woman, severe headaches, to which she had long been liable, ceased when epilepsy commenced, during pregnancy, at thirty-one, but returned when her fits were stopped by treatment at thirty-seven.

Many other instances of this relation could be given, in several of which the migraine was characteristic in its features including visual prodromata.

A still more striking illustration of the connection between the two diseases is afforded by cases in which there is an actual passage of the symptoms of one into the other. Some of the sensory symptoms which precede an attack of migraine bear close resemblance to those which constitute the warning of epilepsy, but differ in their much more deliberate evolution. The sensory disturbance in one hand, which slowly passes up the arm in the course of ten minutes, is like the sensory aura of a unilateral convulsion except that this occupies only a few seconds. In each case such right-sided symptoms may be associated with transient inability to speak correctly. Visual symptoms may precede both migraine and epilepsy, with a similar difference in the time of evolution, but the zigzag spectrum is specially associated with migraine, while simple light or stars precede the epileptic attack. Moreover, hemianopia is common before migraine, and unknown as the aura of epilepsy, which is preceded only by general loss of sight. Yet a transition from one to the other is occasionally met with. A girl had suffered during childhood from severe headaches preceded by "colours before the eyes, red, yellow, and blue;" at fourteen she became subject to epileptic attacks, with loss of consciousness and slight general convulsion, which were preceded by the same visual sensation, much more rapid in evolution. A man became subject, at fifty-five, to a "fortification spectrum" with transverse hemianopia, loss of the lower half of the fields, lasting about a quarter of an hour, but without headache. After a time the duration of the sensation became much shorter, and brief unconsciousness followed; to which was afterwards added slight general convulsion and involuntary micturition. In another patient, a deliberate sensation of numbness in the arm, passing to the lips and roof of the mouth, with a sense of contraction, was followed by severe headache. After these attacks had continued for some years the sensation became much shorter in duration, maintaining the same character, but the headache was replaced by a brief dreamy sensation and a feeling that surroundings were unfamiliar—a characteristic epileptic symptom.

Occasionally attacks of migraine continue after the onset of epilepsy, although seldom with the former frequency. Both affections may co-exist from early life, as in a case of epilepsy since infantile convulsions and migraine from early childhood; the headache always preceded by flame-like lights to one side or the other, and always followed by pain on the side towards which the lights were seen.

CHAPTER VIII.

COURSE OF EPILEPSY.

EPILEPSY may commence in one of three ways. First by minor seizures which occur alone for months or years before there are severe attacks. The attacks of *petit mal* are often at first slight and attract little notice, but become more frequent, until suddenly a severe convulsion occurs. The patient and his friends do not associate the two forms of attack, and it is always necessary to make careful inquiry for the occurrence of minor seizures, antecedent to that which the patient believes to have been the first epileptic fit. It is common, for instance, for a patient to say that he has had fits for a few months only, when attacks of *petit mal* have been occurring for years. I have known such attacks to occur during eight years in one case and six in another, without any significance being attached to them, although one patient often fell. In one instance attention was only directed to the attacks, which at first simply caused the patient, a boy, to drop his book in class, because, later on, he slowly turned round on each occasion. The meaning of the minor attacks is only recognised, as a rule, when they give place to severe convulsive seizures, and often, even then, their significance is not always discerned.

The second mode of commencement is by severe fits recurring at short intervals, without any preceding *petit mal.* The second fit occurs within a few days or a few weeks after the first.

The third mode of onset is with a single severe fit, and no other fit or sign of epilepsy for months and even years, when another attack occurs, after which the intervals gradually become less. Between the last two forms there is every gradation of varying interval between the first and second fit.

SEVERE ATTACKS.—*Interval between the First and Second Fits.*—If an individual suddenly has an epileptic convulsion for which no really adequate cause can be discovered, it is probably the commencement of epilepsy. This is true even if the fit is referred to some excitant, such as an obviously indigestible meal. The actual result is due to a predisposition of the brain, without which only gastric disturbance would result. Whether it indicates epilepsy or not can only be conclusively decided by the occurrence of another fit. Both patient and doctor anxiously ask, If this is epilepsy, when may another fit be expected? After how long a period of freedom may we anticipate that no other fit will occur? No exact answer to this question can be given on account of the great variations in the interval between the first and second fit, the modifying effect of treatment in postponing or preventing a second fit, and because we have no means of ascertaining, on any considerable scale, the frequency with which a single epileptic fit occurs without successors. But some help in forming an opinion may be obtained by comparing the intervals between the first and second fits in a series of cases. It is needless to trouble the reader with the minute details of the statistics; the broad facts are presented by the following statement:—In 14 per cent. the second fit occurred within a week, and in 25 per cent. the interval was between one week and a month. Thus the interval did not exceed one month in about two fifths of the cases. In another 28 per cent. it was between one and six months, and in 10 per cent. it was between six months and a year. Thus in about four fifths of the cases the second fit occurred within a year. These figures only represent the facts as regards severe attacks, and the cases in which minor attacks occurred during the interval would probably lessen very much the proportion in which recurrence was long deferred. They serve at any rate to show that the risk of a second fit remains considerable for at least twelve months after the occurrence of the first fit, and that, although it becomes small after that interval, it does not practically disappear until some years have passed, cases being occasionally met with in which a still longer period elapses, and yet the recurrence is followed by other attacks.

Intervals between Subsequent Attacks.—When the disease is confirmed, the intervals between the severe fits vary greatly, and the variation is still further increased by the effect of treatment.

In order to ascertain the proportion of cases in which the intervals are long or short, I have compared them at the time the patients first came under notice, in a series of 1322 cases. But the fact that many cases had been under treatment up to the time of observation lessens the value of the facts as evidence of the natural course of epilepsy. In some cases the intervals were too irregular to permit of classification, but in 1222 cases the intervals were fairly uniform, not exceeding the limits indicated in the divisions in the following table. Many patients have a series of fits and then an interval, the attacks thus occurring in groups. In this computation the intervals between the fits or groups of fits are taken into consideration, not those between the separate fits which occur together.

Intervals between Fits in 1222 *Cases of Epilepsy.*

	Per cent., omitting fractions.	
Interval not exceeding 1 day . . .	. 11	76 under 1 m.
,, more than 1 day, not exceeding 1 week	. 27	
,, ,, 1 week ,, 2 weeks	. 15	
,, ,, 2 weeks ,, 1 month	. 24	
,, ,, 1 month ,, 2 months	. 10	21—1 to 6 m.
,, ,, 2 months ,, 4 ,,	. 8	
,, ,, 4 ,, ,, 6 ,,	. 2	
,, ,, 6 ,, ,, 12 ,,	. 2	2 over 6 m.

While in a small proportion less than one per cent., the interval exceeded a year.

It thus appears that in more than three quarters of the cases (76 per cent.) the intervals of freedom from fits did not exceed one month. In 50 per cent. the interval did not exceed two weeks, and in 36 per cent. it did not exceed one week, while in 11 per cent. the attacks occurred daily. The interval was between one and six months in 21 per cent., and it exceeded six months in only 2 per cent. It is probable, however, that these figures exaggerate the relative frequency of attacks. A large proportion of the cases were observed in hospital practice, and few patients attend a hospital for attacks which occur only at long intervals.

A very large number of patients stated that they had their fits at intervals of one week, a fortnight, or a month. Little weight can, however, be attached to these statements as evidence of a definite and regular periodicity. When such patients record the dates of the attacks, or are admitted into hospital, it is found that the intervals do not present the alleged correspondence with hebdomadal periods. It is evident that the statements are approximate only, and are suggested by the current divisions of time. A well-marked monthly periodicity is rarely observed except in the cases in women in which the attacks occur in connection with menstruation, either before or after the period.

Many patients have attacks at intervals which vary beyond the limits given in the above table. In these irregular cases the greatest interval exceeded the shortest by from five to forty times. The minimum interval was less than a week in more than half the cases, and the maximum interval was more than a month in nearly three quarters.

Groups of Attacks.—As already stated, the attacks may be isolated or grouped. The former is the more common, but the latter not rare. At periods which vary from one week to several months, series of fits occur, separated by intervals of a few hours, sometimes of a day. The number of attacks varies from two or three to twenty or more. Thus one patient had a series of fifteen or seventeen fits every five days; another had a series of seven or eight fits in the course of a day or two, followed by freedom from fits for a week; another had two to three fits in the course of a night at intervals of two or three months; another had several fits daily for a fortnight and then an interval of several months. The number of attacks occurring in each series often varies considerably in the same individual. Thus one patient, at an interval of a [illegible] sometimes only two fits, sometimes sixteen. Such recurring groups differ, in this customary repetition, and slight severity, from the rapidly recurring attacks which constitute the "status epilepticus" presently to be described.

Changes in the Form of Attack.—Minor attacks, as already stated, may precede severe fits, even during years. They may come on at any time during the course of epilepsy, but especially when the severe attacks become less frequent or cease. Frequently, also, minor attacks, similar to those of idiopathic epilepsy, supervene in cases in which the fits are due to an old organic lesion, especially when this occurred in early life.

Other curious changes in the form of attack are sometimes observed. A child had left-sided convulsions (arm and face), the first soon after a blow on the back of the head by a stone at eight. The convulsions afterwards became associated with laryngeal spasms, which became more and more dominant until the attacks consisted only of laryngeal spasm with loss of consciousness, and almost suffocation, relaxing quickly but not suddenly. These afterwards gradually ceased without recurrence of the previous fits.

Status Epilepticus.—In the cases just described, the patient, as a rule, recovers consciousness in the intervals between the seizures. In rare instances a series of fits occurs in which the patient does not recover consciousness in the intervals, but, while in the post-epileptic sleep, another attack occurs. This has been termed the *status epilepticus* (*état de mal épileptique* of the French). It is a very grave condition. In its most severe form, which has been carefully studied by Bourneville,[*] the intervals between the fits become shorter, the coma deepens, the pulse and respiration become very frequent, and the temperature rises, it may be to 104°, 105°, or even 107°. Sometimes hemiplegia comes on after the condition has existed for several days. The patient may die in a state of collapse, death being apparently due to the violent and almost continuous convulsions, or, the fits ceasing, he may become delirious and present symptoms of meningitis, with rapid formation of bedsores, and may die in this stage. At any period the symptoms may lessen and the patient recover. A large proportion of the cases, however, end fatally. Fortunately this severe degree of the status epilepticus is very rare, except in asylums for the insane. No instance in which death occurred has come under my own observation, although I have seen many examples of a graver degree of the condition, from which the patients have recovered. In most of these the condition was caused by the sudden arrest of the administration of bromides; the cessation of this controlling influence apparently allowed the convulsive tendency to burst forth with intense vigour.

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[*] 'Études cliniques et thermométriques sur les Maladies du Système nerveux,' 1873.

MINOR ATTACKS.—Many patients who suffer from severe attacks, suffer also from minor seizures. The proportion is difficult to ascertain. The occurrence of the slight attacks may elude recognition unless the observer is aware of their common forms, and inquires for them. They are seldom associated with the disease by the patient or friends. Further, attacks are often described as "slight" which present some spasm as well as unconsciousness, because they are less severe than other attacks which the patient has. The attacks of *petit mal* may alone constitute the disease; I have met with one case in which they occurred, without any convulsions, for forty years. They may not only be associated with severe attacks, but also with occasional attacks of post-epileptic hysteroid convulsion, which are constantly mistaken for severe true epileptic attacks. The relative frequency of these combinations, in the cases in which attacks of *petit mal* occurred, is as follows:

Both minor and major attacks	70 per cent.
Minor attacks alone or with hysteroid sequel . .	30 "

Intervals between Minor Fits.—Minor attacks are, as a rule, very frequent in their recurrence. In more than half the cases they occur daily, and often the patient has several each day. I have even known as many as two hundred very slight attacks to occur daily. More frequently there are only two or three, and severe fits occur at intervals of one to four weeks. Occasionally, although minor attacks occur daily, the interval between the severe fits is several months. In about a sixth of the cases with minor attacks no major attacks occurred at any time. When attacks of *petit mal* alone exist they are usually frequent, but if they occur at intervals of many days or weeks they are almost invariably associated with severe fits. The severe attacks may then be more frequent than the slight. Cases are not uncommon in which the severe attacks are frequent, occurring at intervals, for instance, of two, three, four, or seven days, and in which the attacks of *petit mal* occur only at much longer intervals. These cases are, however, rare.

Occasionally there is a connection between the slight and the severe fits. The slight attacks occur only for a few days before, or, much less commonly, for a few days after, the severe seizures. In these cases the interval between the severe fits is usually several weeks. As already stated, the minor fits may occur alone at the commencement of the disease, and then, after months or years of slight seizures, the severer fits may occur. Conversely, if the severe attacks are arrested by treatment, the slight fits often continue.

Time of Attacks.—Another subject which deserves notice is the relative frequency with which fits occur by day or by night, using the terms as synonymous with the sleeping and the waking states. The investigation of this point in 1658 cases has yielded the following conclusions:—The attacks occurred only, or almost only, during sleep in one fifth (22 per cent.); only, or almost only, by day in rather more than two fifths (45 per cent.). They occurred by both day and night in about a third (32 per cent.). Some fits occurred in the night in rather more than half the cases (57 per cent.); attacks occurred by day, with or without attacks in the night, in four fifths of the cases (80 per cent.). In 1 per cent. the attacks occurred only while going to sleep; in ½ per cent. only on waking up out of sleep. In 5 per cent. the attacks occurred soon after waking. A much larger number had fits soon after rising, and in some cases they occurred only at this time. This relation does not seem to depend on the interval after getting up before food is taken, for such patients often had attacks after waking but while still in bed. But it is not wise for the interval after rising before food is taken to be long. This feature seems to be associated with the state of the brain left by sleep, and, in connection with it, we must remember the peculiar tendency, in some cases, to the occurrence of attacks during the process of waking up. We are still ignorant of the state of the brain which underlies sleep. It must be a peculiar condition of the nerve elements in the grey matter, to which the vaso-motor condition is secondary; and we know that the nutrition of the elements occurs chiefly during sleep. We can understand that this may sometimes involve a condition favourable to discharge. It is rare for attacks to occur during the first half of the night; although they may come on as the patient is going to sleep, this is very uncommon compared with occurrence during the process of waking. But cases are met with in which the attacks occur during both transitional states, never during sound sleep or the waking state. Even when attacks occur soon after rising, we can seldom discern a relation to the change of posture, because it is usually some time after this that the attacks occur. Those who are subject to attacks, especially after waking, may also have them after an arm-chair nap in the daytime.

It is somewhat more common for the first fit to occur in the day than in the night—rather more than half commenced in the former, rather less than half in the latter. When the first fit occurred in the day, the subsequent fits occurred only in the day in half the cases, only in the night in one seventh, by both night and day in the rest. When the first fit occurred during the night, the subsequent fits occurred only at night in about two fifths of the cases, both by day and night also in about two fifths, and by day only in the rest. If we may apply to these proportions the doctrine of probabilities, we may say that if a patient who has his first fit in the day has subsequent fits, the probability is as six to one that some of his attacks will occur in the day; the probabilities are equal that his attacks will, or will not, be confined to the day; and they are equal that he will, or will not, have some attacks in the night; while the probability that he will have attacks only in the night is but one in seven. If a patient who has his first fit in the night has subsequent attacks, the probabilities are equal that these also will occur only by night, or that they will occur both by night and by day, being in each case two in five. The probability is as five to one that they will not be confined to the day—that is, in favour of some fits occurring in the night.

Frequently fits which have occurred for a time in one condition, during sleep, waking, or both, change their time. The conclusions deducible regarding these are as follows:—(1) When fits which have occurred only by night begin to occur by day, they commonly continue also during the night. (2) When fits which have occurred only during the day occur during the night, they commonly cease by day. (3) Attacks which have occurred by day and night often cease to occur in the day and continue at night, but very rarely cease by night and continue by day. Of course, these conclusions are liable to many exceptions.

Relation of Attacks to Menstruation.—The relation of attacks in women to menstruation is a subject on which various opinions have been expressed. In one twelfth no attacks occurred at the time of menstruation; in one third there was no difference at these times; in more than half the attacks were worse at the monthly periods. Most frequently they were worse before the period; next in frequency during the period, and much less frequently after the period. Sometimes the period of this occurrence is variable, and may be either before, during, or after menstruation. In two cases fits never occurred at the "periods" when menstruation was regular, but they did when it was irregular.

Pregnancy.—The influence of pregnancy on the occurrence of attacks is various. As already stated, the disease may commence during pregnancy, and may occur only during that state. In one case the fits commenced halfway during pregnancy and continued until the end of lactation, when they ceased, to return during the next pregnancy and period of suckling. Freedom for five years followed, and then they returned spontaneously and persisted. Pregnancy sometimes makes no difference to the disease, but, on the other hand, in the majority of cases they are less frequent during gestation. In one case they occurred then in the night only, although at other times both in the sleeping and waking states. A considerable number of patients have almost or complete immunity during every pregnancy; in one of these a fit occurred three days after each confinement.

As a rule, epileptic women are free during parturition. In one case the first fit occurred three hours after childbirth. If a fit does occur during labour, it has not usually any unfavourable effect.

Acute Disease.—During an acute febrile disease, a patient is usually free from attacks. An exception is scarlet fever, during which they sometimes continue with increased severity. This exception is noteworthy in connection with the influence of this disease in causing epilepsy (see p. 28). The attacks usually return very soon after the cessation of the febrile disturbance, and hence, if it has been necessary to omit the special treatment during the acute disease, it should be promptly resumed. In rare cases such disease is followed by a long period of freedom. A child, with strong inheritance, suffered from fits from two years of age till seven, when they ceased during an attack of typhoid fever, and no recurrence took place until the age of twenty-seven.

CHAPTER IX.

PATHOLOGY.

THE naked-eye appearance of the nerve-centres in idiopathic epilepsy is for the most part that of healthy organs. The slight opacity and thickening of the meninges in some cases of long duration, and the more distinct signs of meningitis and of vascular disturbances seen after death in the status epilepticus, are apparently merely secondary changes. If the patient has died in a fit, the post-mortem appearances are those of the intense venous engorgement which is so conspicuous during life, and they differ little from those met with after ordinary asphyxia. The veins and sinuses of the brain are distended with blood, and so also are the systemic veins and the right side of the heart. Ecchymoses on the surface of various organs have been found after seizures of great severity, and must be ascribed to mechanical congestion. After repeated convulsions fatty changes in the heart and striated muscles have been found (Mott).

Great as is the aid which the microscope has afforded in the investigation of the structural changes in many diseases of the nervous system, it cannot be said to have thrown much light on the nature of idiopathic epilepsy. Of the minute histological changes which have been described, most, if not all, of those which are not common to many other diseases, are simply secondary, the result and not the cause of the violent functional disturbance, or the effect of the repeated passive congestion to which the organs have been exposed. Such are the increased size of the blood-vessels; enlargement of the perivascular spaces, and the presence in them of leucocytes or pigment, relics of recent or old congestion. The yellow pigmentation of the nerve-cells to which importance has been ascribed, is probably without significance. The acute changes in the cortical cells found after death in the status epilepticus (confluence of the Nissl granules) are confined to cases with hyperpyrexia.*

It is more than doubtful whether any importance is to be ascribed to the induration of the cornu Ammonis (*pes hippocampi*), to which so much weight has been attached by Meynert and others. Although changes of trifling character (atrophy and induration with wasting of the cells) have been described as present in one half of fifty cases of epilepsy examined (Bratz, 'Berlin. Gesellsch. f. Psych.,' Dec., 1897), they cannot be regarded as significant, being found apart from epilepsy. All physiological and pathological considerations render it improbable that the lesion has any direct relation to the disease. In the cases of epilepsy which I have examined, the cornu Ammonis was perfectly healthy, and in two cases in which the structure was diseased, the patients had never suffered from convulsion or epileptic symptoms. Nor can weight be attached to the changes formerly described in the sympathetic ganglia. It is difficult to conceive that a disease which may exist for fifty years without the slightest other symptom of disordered action of the sympathetic, can be due to conspicuous alterations in these ganglia.

In considering, then, the pathology of epilepsy, we must seek other evidence as to its nature than that which is afforded by the negative anatomy of the idiopathic disease, and must draw our inferences from the morbid changes in organic disease attended by convulsion, from the results of experiment, and from the facts ascertained by the clinical study of the disease.

It has been already pointed out that the muscular spasm which constitutes the most conspicuous feature of the attack, must be regarded as due to the sudden violent action, the "discharge," of grey matter; and that the sensations which the patient experiences before losing consciousness are due, directly or indirectly, to the same cause—to the commencement of the discharge. Premising this, the subject of the pathology of epilepsy resolves itself, in the main, into four questions:—What is the seat of the discharge which thus produces the symptoms of the fit? Is the seat of the discharge the seat of the disease? How far does such discharge explain all the symptoms of the attack? What is the nature of the morbid change which causes the discharge?

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* Mott, 'Arc [illegible] nty Asylum,' vol. i, p. 493.

What is the seat of the primary discharge? What grey matter is it which thus suddenly stimulates the nerve-fibres and the muscles? It is hardly necessary to remark that the spinal cord acts only as a conductor, and that the overacting grey matter is to be sought for above it, within the skull. Regarding the encephalic masses, we have first the teaching of experiment. On the one hand the researches of Brown-Séquard and Kussmaul demonstrate that in the lower animals convulsions may take origin in the pons and medulla, since they may occur when all other parts of the brain have been removed. Nothnagel has further shown that there exist in the medulla oblongata, adjacent to the centres which regulate the respiratory movements and the state of the vessels, structures capable of giving rise, by their excitation, to general convulsions—"the convulsive centre" as he terms it. But these experimental facts relate only to the lower animals, and the convulsions are not identical in character with those of idiopathic epilepsy (*vide postea*).

On the other hand, of all regional diseases of the brain in man, lesions of the convolutions stand almost alone as a cause of convulsions, and experiments demonstrate that irritation of the cortex in the motor region has the same effect.* The results of experiment seem, indeed, conclusive. A limited gentle stimulation at any point in the motor region causes convulsion limited to the related region, a stronger or a more diffuse excitation causes a convulsion which becomes general. Moreover Ewald† has found that excitation of the auditory and visual regions causes characteristic epileptic convulsions, in harmony with the clinical facts of attacks which begin by an auditory or visual aura. These centres must be intimately connected with those for motion, as illustrated by the energetic motion in animals caused by an alarming sight or sudden sound.

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* Hughlings Jackson, Ferrier, Luciani, and many others.

† 'Neur. Cent.,' 1898, p. 619.

The experiments of Nothnagel have given rise to many others, in order to ascertain the precise origin of the convulsive spasm. The investigations of Zulien suggested that clonic spasm had its origin from the cortex, tonic spasm from a centre in the pons. This view is inconsistent with the results of simple observation of convulsions. The most distinct feature of the spasm is the gradual transition of the tonic to the clonic spasm, by remissions, at first fine, but becoming coarser, until they amount to distinct intermissions. It is impossible to doubt that both are forms of the same process and proceed from the same seat. That tonic spasm may be produced by stimulation of the pons may be a fact, but it has no bearing on the origin of that of epilepsy.

The subject has been thoroughly investigated by Bechterew and his pupils.* He confirms the notorious fact that convulsions are excited by electrical stimulation of the cortex, and, according to its degree, are local or general. Further, the convulsion excited by prolonged stimulation may consist either of tonic spasm or of tonic which passes into clonic spasm, followed by coma, as in a typical epileptic fit. The result is especially typical if the motor centres of both hemispheres are simultaneously excited. But the same result follows if one centre has been previously excised. If both cortical centres have been removed, the stimulation of the white substance causes only tonic spasm ceasing with the application. The effect of injection of substances that cause convulsion, as absinthe or cinchonine, indicates that convulsion *may* be generated from the mesencephalon or lower structures, since it was produced after removal of the hemispheres, but with only a slight degree and irregular form of clonic spasm, while, after partial removal of the cortical centres, the clonic spasm was slight in the parts corresponding to the centres removed, but the tonic spasm was equal everywhere.

Although these facts confirm the view that convulsion, especially tonic, may be generated from the mesencephalon, they constitute no evidence that the attacks of ordinary epilepsy arise there. Moreover, when disease of this region causes convulsions, there is often or generally a high probability that it does so by an upward influence on the cerebral cortex. Bechterew found that the convulsions produced in guinea-pigs by the puncture of the pons with a needle, ceased if the cerebrum was separated from the pons. The conclusion he reaches as the result of his laborious and important investigations is that, in epilepsy, the convulsions are due to discharge in the cerebral cortex, and that, although the basal parts may give rise to convulsion in injury and poisoning, this is chiefly tonic; and when the convulsion is distinctly epileptiform, it is undoubtedly due to the cerebral cortex.

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* Bechterew, 'Neurol. Centralbl.,' 1895.

The teaching of pathology is clearly, as Wilks long ago insisted, that epileptiform convulsions have, in most cases, their origin at the surface of the brain. Organic epilepsy is found always to be due to cortical disease, to active irritation, as by a tumour, or to an old stationary lesion, especially traumatic or other softening. The facts illustrating this have been already mentioned. They justify the conclusion that epilepsy is a disease of the cerebral cortex.

Pathology thus points to a definite conclusion, to which experiment is less opposed than may be assumed. From clinical observation we may gain other important information. The study of the modes of onset of convulsions is specially important as supplying evidence of the structures in which the discharge commences. The commencement in the limbs is, when deliberate, by movements which we only know to be differentiated in the cortical centre. Equally, or even more significant, are the auras referred to the special senses, or consisting in mental processes. We have seen that, of all the fits which begin so deliberately as to allow the patients to be conscious of the onset, a special-sense warning is present in a fifth—that is, the discharge, as far as we can ascertain, commences in a special-sense centre of the cortex. It has been proved, moreover, that stimulation of the auditory or visual centre in an animal may excite a convulsive attack.* Hence the conclusion seems inevitable that the discharge in such cases commences in the hemispheres. It is equally clear that an attack which begins with a psychical aura cannot commence with a discharge in the medulla oblongata. Such an attack we can only conceive as commencing in the highest of the cerebral centres.

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* Ewald, 'Neur. Centralbl.,' 1898, p. 619.

We may therefore conclude, with Hughlings Jackson, that the teaching of pathology, if inconclusive alone, is right in its indications, and that, in at least most epileptic attacks, the process of the fit commences in the cerebral hemispheres. The conclusion thus reached derives additional strength from the experimental demonstrations, already referred to, that epileptiform convulsions may be excited by irritation of the motor region of the cortex. The case of epilepsy recorded by Oebeke (see p. 103) is of special significance, in that convulsions, formerly general, were arrested on one side by a lesion in the central ganglia of the opposite hemisphere, demonstrating that the discharge in idiopathic epilepsy may proceed from the cortex of both hemispheres by their simultaneous activity. I have met with a similar case; frequent general convulsions occurred from two years of age until fourteen, when an attack of right hemiplegia came on with transient aphasia. Six years later there was still considerable weakness of the right limbs with inco-ordination and mobile spasm of the hand. The fits had continued, but ever since the attack of hemiplegia they had been limited to the left side, and commenced by deviation of the head to the left.

If the significance of these facts is clear, and most epileptic fits are to be regarded as the expression of a morbid process in the cerebral cortex, the question arises, are we not justified in assigning all idiopathic convulsions to this situation?

There is one group of auras which may be regarded as supporting the view that the fits which they initiate take origin in the medulla; the warnings that consist of a sensation referred to the functions of the pneumogastric, consisting of epigastric sensations rising to the throat, choking, dyspnœa, palpitation, commence in this part. The pneumogastric nucleus and respiratory centre are believed to be in close proximity to, and connection with, the supposed convulsive centre, and it is readily conceivable that a commencing discharge in this situation may lead to disturbance of the respiratory and pneumogastric centres before it causes general convulsions. But on this point other facts must be taken into consideration. The phenomena of conscious sensation make it certain that the whole body, with all its functions, is represented in the cerebral hemispheres. When a discharge originates in certain parts of the hemispheres, the central structures connected with the vagus, or representing the respiratory processes, may be the seat of the first discharge, or may, at least, be the most sensitive to the commencing disturbance, and be the channel through which the consciousness is first impressed. We must remember that these central representatives of the vagus are the most readily disturbed in emotion, such as fear. Thus the early affection of these centres, which we have just considered to be possible in a discharge originating in the medulla, is equally possible in a discharge originating in the cerebrum. No weight can therefore be placed on the pneumogastric aura as evidence that the discharge originates in the medulla. This conclusion is supported by cases which show that this aura is sometimes felt in attacks which certainly commence in the hemispheres. As already stated, this pneumogastric aura may be associated with special-sense warnings even of high elaboration.

Cases are very few which afford any clinical evidence that the discharge commences in the medulla. It is possible, however, that, in cases of reflex convulsions, especially in infancy, the process occurs in this situation. Almost the only example of idiopathic epilepsy which has come under my notice in which the symptoms suggested that the discharge originated there was the patient whose case is mentioned on p. 109, in which passive movements of the trunk, as slight flexion of the spine, invariably caused an epileptic fit, attended by loss of consciousness. Although there was no epileptogenic zone, the attacks remind us of Brown-Séquard's epileptic guinea-pigs, in which the convulsions certainly did not originate above the pons. But in such a case it is probable, from the loss of consciousness, that the result was produced by an upward influence on the cerebral cortex.

The conclusion, then, is that the convulsions in idiopathic epilepsy commence by the discharge of the grey matter of the cortex. This brings us, however, to the second question: Is the seat of the primary discharge the seat of the disease? If, by such discharge, all the symptoms can be accounted for, we have no need to go further, and the theory that any other pathological process underlies the phenomena must rest upon its independent evidence. To learn, therefore, whether the seat of the discharge can be regarded as the seat of the disease, we must consider, first, whether there are any symptoms for which the discharge of grey matter, which causes the aura and the convulsion, cannot account; and, secondly, whether there is direct evidence of other morbid action.

It was formerly held that the discharge itself, though originating in the hemispheres, is due to vaso-motor spasm affecting particular arteries, and exciting the discharge by causing local cerebral anæmia. Another theory ascribed the convulsion to the discharge of a convulsive centre in the medulla, and the loss of consciousness to arterial spasm in the hemispheres due to the action of the vaso-motor centre in the medulla.

Is there any evidence that vaso-motor spasm in the brain causes loss of consciousness, or any of the symptoms of the attacks? The evidence may be put, if somewhat baldly, yet I think accurately, thus:—Epileptic attacks are accompanied, at the onset, by pallor of face. Pallor—that is, anæmia—of face is evidence of anæmia of brain. Anæmia of brain, as Kussmaul and Tenner have shown, may cause loss of consciousness and general convulsions. The arteries of the meninges (and probably also of the brain substance) may be made to contract by irritation of the sympathetic in the neck. Therefore loss of consciousness in epilepsy is due to anæmia of brain, the result of vaso-motor spasm; and, some would add, the convulsions themselves are due to the same cause.

The gaps between the steps of this reasoning are considerable. Moreover the first two statements are themselves not true. Epileptic attacks are often accompanied by no pallor of face, and this is true most conspicuously of many attacks of *petit mal*. Further, the assumption that vaso-motor spasm of the face is evidence of vaso-motor spasm in the brain is not warranted. Surface vessels have no necessary correspondence in their condition with those of deeper structures. We do not infer that the brain is congested whenever the face blushes, and why should we assume that the brain becomes anæmic whenever the face becomes pale? We have seen that a discharge in the brain may cause a contraction of the peripheral vessels. Pallor of face is conspicuous as part of the ordinary phenomena of cardiac syncope, but convulsion is not. Although the discharge may impress the consciousness first through the pneumogastric centres, it is certain that cardiac failure is not the *ordinary* cause of loss of consciousness in epilepsy; loss of consciousness may occur without either failure of the pulse or pallor of the face.

Thus between the fact that profound anæmia of the brain and medulla oblongata will cause both loss of consciousness and convulsion, and the theory that these are commonly so caused in epilepsy, there is a gulf over which no bridge has yet been placed.* It is a gulf, moreover, which is widened by every addition to our knowledge of the clinical history of the disease.

The clonic spasm of the epileptic fit is developed from the tonic spasm, first by slight remissions, gradually increasing to complete intermissions. The clonic stage corresponds to the cyanosis which results from interference with respiration, and observation of fits suggests that the venous congestion induces the change, as Axenfeld and A. Foville have asserted, by interrupting and ultimately arresting the spasm which before existed. The fixation of the chest in an epileptic fit simply does in an intense manner that which we do in a slighter manner when we arrest an hysterical convulsion by closing the mouth and nose. The view that the tonic spasm is only clonic spasm compressed, that the clonic spasm is only the tonic spasm spread out, is supported by the fact that in attacks which begin slowly the slight initial spasm is almost always clonic, and as the discharge spreads and becomes more intense the spasm becomes tonic. In local epileptiform seizures the spasm is often clonic throughout, which is also in accordance with the physiological fact that tonic spasm consists in, so to speak, superimposed clonic spasm.

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* "Although experimental investigation has thrown much light upon the dark places of experimental pathology, it has brought with it some confusion into the region of practical medicine. There are analogies and close relations between the convulsive phenomena of rabbits which are bled to death, of guinea-pigs with their spinal cords half sundered, and the convulsive paroxysms of epilepsy; but the cases are not identical, and although experiment may elucidate the mechanism of the attacks—some parts of which may be the same in the two cases—it may leave untouched the real pathology of the disease, the clinical history of which is peculiar to itself and more or less widely different from those of the several paroxysms artificially induced."—Reynolds, 'Epilepsy,' p. 250.

Moreover, the peculiar functional disturbance with which many fits begin, often precise and uniform in all attacks, is a symptom which cannot be explained by so random an influence as arterial spasm.

The conclusion, then, is that all the phenomena of the fits of idiopathic epilepsy may be explained by the discharge of grey matter; that the hypothesis of vascular spasm is as unneeded as it is unproved; that there are no facts to warrant us in seeking the seat of the disease elsewhere than in the grey matter in which the discharge commences; that this is in most cases within the cerebral hemispheres, generally in the cerebral cortex, although possibly in some instances lower down, even in the mesencephalon in the case of convulsions of untypical character.

While we can thus refer the discharge of epilepsy with confidence to the cortex of the brain, it is otherwise with the precise elements of the cortex in which it originates. Until lately no doubt was felt that the nerve-cells are the source of nerve energy, that from them the nerve impulses proceed alike in health and in disease. But the recent change in opinion regarding the constitution of the nervous system, the discovery that it consists of discontinuous neurons, suggests that this opinion must be modified. The fact that each neuron consists of a group of fibrillæ, associated in the axis-cylinder process, separating at its termination, passing uninterruptedly through the nerve-cell, and separating in its branching processes or dendrons, to terminate by free extremities in the spongy grey matter, involves a more complete change in our conceptions than is as yet everywhere recognised. We have to regard the nerve-cell as the vital centre of the neuron, through its nucleus and protoplasmic prolongations, but without relation to the production of nerve energy, if the nerve-fibrillæ pass continuously through its substance. We must look to the terminations of these in the spongy substance for the origin of the impulse, just as it originates at the periphery (skin, etc.) in the extremities of the separated fibrillæ. In the spongy substance the renewal of nutrition can take place readily from the free access of plasma, as it can scarcely do in the closed nerve-cell. Function must be excited by the passage of impulses to neighbouring dendrites which terminate in proximity. But there must be a close relation between the fibrillæ of associated neurons to permit voluntary action of many muscles, and there must also be a readiness for energetic action which may conceivably permit apparently spontaneous discharges.

Considerable as may be the difficulties involved in this conception of the infinite subdivision of the discharging elements in the spongy substance, they are scarcely less if we regard the nerve-cells as the source of discharge. Even in their case the multiplicity of the discharging elements is so vast that the difficulty of conception remains almost the same, and yet from such difficulty there is no escape.*

Physiological knowledge does not enable us to discern clearly the nature of the process of "discharge." The precise nature of nerve force and of its conduction are still matters of discussion. We cannot doubt, however, that the sudden morbid production of nerve energy in disease occurs by a process essentially the same as that which permits its release in health. All that we are able to discern indicates that this process depends on chemical union between the molecules of the nerve structures and the oxygen conveyed to them from the blood by the plasma with which they are bathed. Within the complex molecules of the nerve substance the atoms are in delicate equilibrium, kept apart by a high degree of interatomic motion, which only needs the added motion of a "stimulus" to release them and permit the closer union with the oxygen from which simpler compounds result, with less intra-molecular motion. The balance escapes as nerve force, apparently putting down the conducting tracts, continuous series of molecular combinations, by a process analogous to that by which it arose—a process of chemical action under the influence of vital conditions, of explosive rapidity, but strictly limited in range. Electrical changes attending conduction are probably collateral, and not direct indications of the nature of the process, although the future may perhaps show that they are related to it in ways of which we have as yet no conception.

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* Recognisable changes in the cortical dendrites have been described as the effect of toxic doses of bromide, the agent that chiefly controls epilepsy.—Wright, 'Brain,' Summer No., 1898.

If we recognise, as apparently we must do, that chemical processes, in the vital tissues of the highest elaboration, underlie normal nerve action, we must also recognise the extreme suddenness with which they may be produced (as in the instant reflex action, or the energetic movement to escape perceived danger). The suddenness of the discharge in epilepsy scarcely transcends that of normal action, at least not in a degree to make it difficult to refer both to the same process. The difference lies in the occurrence of the morbid discharge without the normal stimulus. We must assume an abnormal state of chemical nutrition, whereby the molecules are more readily detached, and their atoms yield with undue facility to the attraction of the adjacent oxygen. It may be that this release is effected by a stimulus too slight to be perceived, or the process of morbid nutrition may proceed to the degree of dissociation, so that a spontaneous separation and simpler interatomic union takes place. Once set up, the motion released will act as a stimulus on adjacent molecules and spread in the nerve substance with the explosive rapidity which vital nutrition seems to permit.

All functional activity involves a breaking up of molecules and their renewal from the nutritional constituents of the plasma. But they are not renewed in precisely the same manner. There is a difference in constitution, distribution, and relation, which, minute though it is, facilitates a repetition of the same process. It makes the recurrence of any discharge more facile, and this in a degree proportioned to the frequency of its occurrence. It is this tendency which normally permits habitual acquisitions, and renders such a disease as epilepsy self-perpetuating. The tendency is only to be prevented by long-continued restraint of the morbid process, by which the nutrition is at last fixed in normal stability.

The process of inhibition, which plays so prominent a part in many minor attacks, and in the initial stage of many severe seizures, seems at present to baffle our efforts to explain it. It was formerly regarded as the result of an increase in that "resistance" in the nerve-centres which normally controls and limits nerve activity. The resistance was supposed to be a function of nerve-cells related to, but distinct from, that which causes their discharge. But, when scrutinised, this is merely a translation of the phenomena observed into terms of nerve physiology. The fact of "inhibition," of arrest of action, is certain; but its nature is not elucidated by its description as "increased resistance." We need to have some conception of the process by which activity is permitted and prevented, and of that we have at present no discernment. This is seen if we consider the fact that the seat of discharge, the structures whence nerve energy is released to traverse the fibrils, may be, perhaps must be, the extremities of the dendrites, of the branching processes which end in the spongy grey matter, and for the most part conduct towards the cell, and for the most part also through it. If this is true, we must look to the structure and function of the mysterious substance in which the dendrites end for the explanation of these phenomena. Without accepting the doctrine of amœba-like movements of the dendrites, bringing them now into proximity to the nerve-endings from which they are stimulated, and now withdrawing them from the influence, we can yet conceive that changing relations may occur, which give rise to the phenomena observed. We know nothing of the functional characteristics of this spongy substance, but can conceive that they may be such as to subserve all the conditions necessary to explain the facts.

The phenomena of hysteroid seizures seem to point to an instability of grey matter of different seat and perhaps different kind. The excessive violence of the muscular spasm in that disease is comparable only to the convulsions of epilepsy and tetanus. The phenomena appear due to the more deliberate and sustained overaction of centres which are concerned in specialised, co-ordinated movements, and in emotional manifestations. The fact that these seizures often succeed attacks of epileptic *petit mal* has been regarded by Hughlings Jackson as evidence that the hysteroid convulsion (like the automatic action, p. **141**) is always due to the release of lower centres from the control of the higher by the temporary discharge of the latter. He has urged that an actual discharge of grey matter can only give rise to inco-ordinate "coarse" effects. That co-ordinated convulsive action may succeed attacks of true epilepsy, is beyond question; but it is also beyond question that the co-ordinated phenomena are not always preceded by epileptic symptoms, and, on the other hand, it seems certain that co-ordinated movements may result from a true discharge such as occurs in epilepsy.

It has been already suggested that even for the cases in which this symptom succeeds a true epileptic seizure the hypothesis of "loss of control" scarcely affords an entirely adequate explanation. We can discern no difference between epileptiform attacks which are, and those which are not, followed by such symptoms. Hence their occurrence must depend upon some other condition, a condition pertaining to the nervous organisation of the individual, and not to the form of attack. This is supported by the fact that cases, in which epileptic attacks are succeeded by hysteroid convulsion, present preponderant characteristics in age, sex, causation, and the conditions under which the fit occurs. In these respects the patients resemble those in whom such attacks of hysteroid character occur without any preceding epileptoid symptoms. These facts make it probable that there is in these epileptics, in addition to the instability of nerve-tissue which causes epilepsy, also the instability, whatever it may be, which gives rise to hysteroid attacks. "Loss of control" may be the immediate mechanism by which this instability is thrown into operation, but it will not alone account for its existence, and is no necessary condition for its manifestation. Nevertheless, in some patients, the hysteroid tendency may be so slight that it never manifests itself spontaneously—never except during the post-epileptic state.

Such forms of attack, in which the convulsive phenomena are violent in degree and disorderly in sequence, are in the lowest grade of co-ordinated phenomena, while the highest are those forms of involuntary action which we call automatic. In these, complex actions are performed, sometimes with considerable skill, of which the patient is subsequently entirely unconscious. Hughlings Jackson has maintained that these are really post-epileptic phenomena. That they are commonly such, all must, I think, agree, but analogous symptoms sometimes initiate attacks. The vision of an old woman making ugly faces, which one patient had sometimes as the aura of a fit, sometimes as an attack of *petit mal*, must be regarded as a co-ordinate discharge in—an automatic action of—the centre for visual ideas. The remarkable aura which I detailed at length (p. 76) suggests the same explanation, and so do many others that have been mentioned. I have seen a patient suddenly, while being watched, without the slightest pallor, hesitation, or any symptom to indicate a previous "discharge," proceed to empty his pockets, take off his coat, or do some action which characterised, and perhaps constituted, his attack of minor epilepsy.

With regard to these attacks of post-epileptic automatism, the same difficulties face us as in the case of the co-ordinated convulsion. We have the facts that in one individual an attack may always be followed by automatic action, and in another, similar attacks may never be so followed. This suggests that there is, underlying the phenomenon, a condition of the centres on which it depends. Were it not for this condition, the epileptic attack would not lead to it, and this condition may, in rare cases, cause its primary manifestation.

So too with regard to the violent attacks of automatic action which are termed "epileptic mania," and which, as we have seen, while usually post-epileptic, apparently sometimes precede epileptic fits, and sometimes occur apart from any recognisable fit. We know too little of the ultimate nature of cerebral action to enable us to venture on a pathological analysis of such phenomena with any sense of security.

A special toxic character of the blood after epileptic fits has been said to exist by some observers. Others have been able to discover no evidence of it. The existence of such a state is not sufficiently established to make it of practical significance.

CHAPTER X.

DIAGNOSIS.

THE problem of the diagnosis of epilepsy includes (1) the recognition of the occurrence of attacks; (2) the distinction of the attacks from other paroxysmal affections with which they may be confounded; (3) the distinction of hysteroid from epileptic attacks; (4) if there is hysteroid convulsion, we have to ascertain whether it is simple or consecutive to an epileptic seizure; (5) if the convulsion is apparently epileptic, we have to consider whether it is due to reflex irritation, to blood-poisoning, or to organic brain disease, active or latent, and also whether it is simulated, before we can conclude that it is the manifestation of idiopathic epilepsy.

(1) *Recognition of the Occurrence of Attacks.*—Severe convulsive attacks are such obtrusive phenomena that there is little difficulty in ascertaining their occurrence, except in the cases in which they come on only when the patient is asleep and alone. If the patient is not awakened by the commencing attack, and sleeps on when it is over, he may be entirely unaware of its occurrence. Usually, however, there are some subsequent indications of the attack. The tongue is sore on one side from having been bitten; there is a little blood upon the pillow; or there are small ecchymoses upon the face of the patient, or an extravasation beneath the conjunctiva. But I have known a patient to have convulsive attacks at night every few months for eighteen years, without being aware of them. His tongue was occasionally found to be sore in the morning, but the significance of this was not suspected until an attack occurred in the daytime. The

passage of urine into the bed may be another sign of attacks, but sometimes the only indication is that the patient wakes with a headache and a general feeling of fatigue. In one recorded case, nocturnal fits were only discovered by a dislocation of the shoulder.

Minor attacks are very often unrecognised by patients or their friends; if discerned, their nature is often unsuspected. Sometimes their occurrence is known only to the patient; they consist of a sensation only, with no symptom to attract the attention of others. A strange feeling, often undescribable, sweeps over the patient and is gone. On the other hand, they may be unknown to the subject and perceived only by the friends, because there is a moment's loss of consciousness so sudden and complete that the patient is unaware of it. He may even continue some automatic action, as walking. Even when a normal state is regained slowly, the process of gradual return may leave no effect on memory, or there may be only a slight sense of confusion, the significance of which is not suspected.

The chief features of these attacks, by which their occurrence may be known, have been already described. But it is important to remember how frequently those are overlooked which consist only of a subjective sensation. Extremely various as are the characters of these sensations, they all present the features of sudden occurrence, usually without any excitant, of momentary duration, and of sudden cessation. It is important to ask whether any sensations have been felt of any kind, coming on when the patient is still, lasting for a moment, and then passing away. But such a general question does not always suffice to ascertain the occurrence of minor attacks, which may, nevertheless, be frequent. It is necessary to ask definitely for some of the more common forms. It is not found that "leading questions" involve the risk of error, in these cases, which often attaches to them. Patients are anxious to deny, rather than to admit, the impeachment submitted to them. The sensations that should be asked for are—sudden darkness from momentary loss of sight, sudden lights or stars before the eyes, a momentary noise in the ears, a sudden sense of strangeness, of being in an unfamiliar place, a brief "dreamy" feeling, a momentary epigastric sensation, or momentary giddiness. With the exception of the last these sensations are not described unless they have been actually felt. The exception of "giddiness" is only partial because the term is so often applied to mere mental confusion when there is no sense of vertigo. The nature of the feeling should therefore always be ascertained. When the sensation is actually one of turning or of falling we have to remember that this has other causes. It occurs from aural disease (a question that will be considered presently), and also from weakness and anæmia.

Epileptic vertigo is usually attended by a more distinct sense of mental confusion, otherwise its features resemble the simple form except for the important fact that it occurs always or generally when the patient is still. Simple vertigo is almost always excited by movement, rising from the sitting posture, or suddenly turning. Yet momentary giddiness on movement is sometimes connected with epilepsy, for it may be experienced by a patient during the day or two before or after a severe fit. The spontaneous character of the giddiness is therefore of more significance than its excitation. The distinction of epileptic from aural vertigo will be presently considered.

(2) *Distinction of Attacks from other Paroxysmal Symptoms with which they may be confounded.*—Severe fits are so characteristic that their nature is usually at once recognised. It is very different with the minor attacks which give rise to some definite diagnostic question.

Syncope.—The simplest form, characterised by transient loss of consciousness, lasting for a moment, and passing away, to leave the patient just as before, is constantly regarded as a faint, signifying no more than an ordinary syncopal attack.

The distinction from fainting attacks rests, first on the absence of obvious exciting influences. Syncope occurs in weakly persons, and under the conditions of mental emotion, over-exertion, heated rooms, during diarrhœa, severe abdominal pains, etc. The "faints" of epilepsy occur in strong as well as in weakly persons, and come on at all times and under all circumstances. Syncope is preceded as well as accompanied by pallor and by failure of the pulse. In the epileptic faint the colour of the face is commonly unchanged at the onset and the pulse maintains its normal character. In epilepsy the loss of consciousness is often quite sudden; in syncope a feeling of faintness, often accompanied by loss of sight or nausea, with perspiration, usually precedes the unconsciousness. But the absence of a sense of faintness is of greater value as an indication of the epileptic nature of the attack, than is its presence as evidence that the attack is syncopal, since some minor epileptic seizures are preceded by an aura which is described in similar terms. In epilepsy there is often also some other aura, such as have been described at length. Some of these, as the epigastric sensation, a visual sensation, or a sudden and peculiar mental feeling (*e. g.* a sudden sense of "strangeness"), do not occur in syncope, and so are of considerable diagnostic importance. Giddiness, if definite vertigo, also suggests epilepsy rather than syncope. But indefinite "dizziness," palpitation of the heart, nausea, cephalic sensations other than pain, and failure of sight, are of little diagnostic significance. The stage of complete loss of consciousness is much briefer in epilepsy than in syncope. Micturition, which may occur in the former, is unknown in the latter. Consciousness may be regained more suddenly in epilepsy than in syncope. It may be gone and back again in a second or two. In either, the normal condition may be slowly regained, but the return is through physical faintness in syncope, and mental confusion in epilepsy. After the latter some automatic action may be performed, a phenomenon never observed in cases of simple syncope. In the vast majority of cases, attention to these features will enable the diagnosis to be made without difficulty. In a large number of cases of minor epilepsy the patient has other, more severe seizures, which assist the diagnosis.

Aural Vertigo.—The form of definite vertigo which occasions the most serious diagnostic difficulty is that which is due to labyrinthine disease, called aural or auditory vertigo, or, in the most severe forms, "Menière's disease." The last, very intense varieties, are always distinct in nature. If there is risk of their confusion it is with organic brain disease, not with epilepsy, from which it is the slighter forms of special character that have to be distinguished.

Aural vertigo is generally associated with obtrusive symptoms of labyrinthine disease, tinnitus and nerve-deafness. The latter may be slight or considerable, recent or of long duration. The tinnitus is constant and is not limited to the actual onset of the attack, as when it is the warning of epilepsy. The onset of aural vertigo may be quite sudden, but it rarely ceases suddenly; it passes off gradually, usually lasting for hours, and if vomiting follows, this is distinctly due to the intensity of the giddiness. The vertigo of epilepsy ceases as suddenly as it came on; it is often attended by loss or impairment of consciousness, and the attack passes off by mental confusion, quickly lessening, instead of by actual giddiness.

A special difficulty, however, is presented by some cases in which attacks, really labyrinthine in origin, present a misleading resemblance to those of epilepsy. They are cases in which there is considerable cerebral instability, and the attack bears no close relation to the type. The onset of the attack is absolutely sudden; indeed, the patient may fall with the sensation that he has received a violent blow on the head, but it is a sense of violent momentum received, not of any surface impact. There is often impairment of consciousness, sometimes amounting to brief actual loss. We can understand that this may result from pure vertigo if we remember that normal consciousness consists largely in a normal perception of our relation to the environment, and how profoundly this is disturbed in vertigo. The suddenness of the onset must depend on the fact that the centres concerned in the maintenance of equilibrium, and perhaps the related sensory centres of the brain, have been brought into a preceding condition of instability, and the actual attack is probably due to a process analogous to the discharge of epilepsy. Whatever is the chief seat of the disturbance, its perception must be through the cerebral hemispheres, and it is not difficult, therefore, to understand the occasional occurrence of actual loss of consciousness. Such cases also are distinguished by the indications already mentioned, the vertigo which succeeds the attack, the labyrinthine symptoms, and the fact that the patient has other slighter attacks of more typical character.

Another, but rare, difficulty in diagnosis is presented by cases of epilepsy in which the first disturbance of consciousness is due to discharge in the region of the brain disturbed in aural vertigo. The centres concerned in the maintenance of equilibrium are intimately connected with those for the pneumogastric (through which consciousness is so often first disturbed in epilepsy), as well as with the central representation of the auditory nerves. It is therefore not surprising that in some patients the symptoms of epilepsy should bear a very close resemblance to those of aural vertigo. An auditory and pneumogastric sensation may be combined with vertigo as the warning of an epileptic fit. This was the case, for instance, in a man aged twenty, who had suffered for six months from attacks which commenced with a sensation at the epigastrium "as if it were turning over and over," followed by a "confused feeling" in the head, making him take hold of some support to avoid falling. This was succeeded by a whistle in both ears like a "distant railway whistle going through his head." Consciousness was lost and the convulsion was severe. The patient had also minor attacks, consisting only of the aura and momentary loss of consciousness.

It must be remembered that a patient may suffer from both epilepsy and auditory vertigo. There is nothing mutually exclusive in the two morbid states. Indeed, their concurrence is intelligible if we consider, as I think we must, that the occurrence of vertigo, *i. e.* the disturbance of the equilibrial centre, which is produced by the ear disease, is facilitated by any primary instability of the nerve-centres; and such instability may be part of the general defective nutrition of the brain of which the epilepsy is a result. Much care is of course necessary in the recognition of the presence of two diseases which have many symptoms in common, but in several cases which have come under my notice the evidence of the co-existence of the two affections has been clear. A woman suffered from distinct epileptic fits from forty-seven to fifty-three, when they ceased. At sixty-three she became liable to paroxysmal vertigo—a sensation of falling backwards and towards the right, without loss of consciousness, and followed by vomiting. There was complete loss of hearing through the bone (watch and tuning-fork). The attacks of giddiness were often induced by movement, and especially by sudden rotation of the head to the right, not to the left. After she had suffered from these attacks for a year, the convulsive fits recommenced, the vertiginous attacks continuing. Again, a girl of twenty-eight had been epileptic for several years; the fits, many of which were witnessed, being severe. She also suffered from almost constant giddiness, worst after rising in the morning, and varied by severe paroxysms of vertigo, often produced by movement, in which she seemed to fall backwards and to the left. She had constant noise in the ears, and loss of hearing through the bone. Another patient, with characteristic aural vertigo, suffered also from attacks of minor epilepsy, in which loss of consciousness was preceded by an olfactory aura and epigastric pain.

Neuralgia.—Cases of minor epilepsy of which the aura is a sudden pain in the head may be mistaken for neuralgia; but the occurrence of transient loss of consciousness after the sudden pain is sufficient for the diagnosis.

(3) *The Distinction of Hysteroid from Epileptic Attacks.*—If a patient suffers from attacks of distinct convulsion, the next question is, are the attacks epileptic or hysteroid? If a fit can be witnessed, it is rarely that any diagnostic difficulty will present itself. As a rule, the characters of the convulsions are distinct enough. The violent tonic and shock-like clonic spasm of the typical epileptic fit, with its complete unconsciousness, cyanosis, and brief duration, are wholly unlike the wild co-ordinated movements, perverted consciousness, talking, and even biting of the hysteroid seizure. Other distinctive characters of the latter are the long-continued tonic spasm, often opisthotonic, the quivering of the eyelids, the quick clonic spasm, maintaining the same

rapidity throughout, the rolling up of the eyeballs when exposed, and especially their convergence, and the long duration of the convulsion. These attacks have been already described. It is important, moreover, to remember they suggest irresistibly to the friends the need for restraint. The patient has to be held down. This is never the case in epilepsy, in which the patient has only to be kept from hurting himself in the clonic spasm.

Excitation by emotion, although a common feature of hysteroid attacks, is also met with in true epilepsy (see p. 108), and too much reliance must not be placed on it. The emotion which excites the hysterical attack is usually profound, at least in its apparent character, whereas that which excites an epileptic fit is more frequently some trivial start.

An important diagnostic feature is that simple hysteroid attacks do not recur frequently during years, and especially, do not begin in childhood. Careful inquiry should be made as to the form of spasm; there is struggling in the hysteroid, jerking in the epileptic attack. Another indication, which has been already mentioned, is whether the patient has to be held down or merely prevented from hurting himself or herself. The former points to hysteria, the latter to epilepsy. Micturition during the fit and tongue-biting are conclusive epileptic symptoms, since hysterical patients never bite the tongue. They may bite their lips and sometimes try to bite other people. These diagnostic points are given in the following table. In their use it should be remembered how often the two forms are combined (see the following section), and that automatic action, after a simple slight epileptic fit, is also liable to mislead.

Diagnostic Characters of Epileptic and Hysterical Fits.

	EPILEPTIC.	HYSTEROID.
Exciting cause .	Rare.	Often emotional disturbance.
Warning . .	Any, but especially special senses, unilateral, or epigastric aura.	Palpitation, malaise, choking.
Onset . . .	Sudden.	Sometimes gradual.
Scream . .	At onset, strange in sound.	During course, quasi-volitional.

	EPILEPTIC.	HYSTEROID.
Convulsion .	. Rigidity, followed by "jerking," rarely rigidity alone.	Rigidity or "struggling," throwing limbs and head about, fighting, kicking.
Biting .	. Tongue.	Lips, hands, or sometimes other people and things.
Micturition .	. Frequent.	Never.
Defæcation .	. Occasional.	Never.
Talking .	. Never.	Occasional.
Duration .	. A few minutes.	Often half an hour or longer.
Restraint .	. To prevent accident.	To control violence.

(4) *Distinction of Simple from Consecutive Hysteroid Attacks.*—If there is evidence that the described convulsion is hysteroid, we have to ascertain whether it is simple, or whether it is consecutive to an epileptic seizure. The frequency of this sequence has been already illustrated. It is important, however, to bear in mind the fact that some patients who have epileptic attacks followed by hysteroid convulsion, have, at other times, hysteroid attacks which do not succeed epileptic fits.

Cases which give rise to some difficulty are those in which the hysteroid sequel is brief, and the whole attack does not exceed the duration of an ordinary epileptic fit. In one seizure of the kind which I witnessed the patient suddenly fell backwards, absolutely unconscious, and would have fallen with violence had he not been caught. The limbs and trunk passed instantly into violent strained tonic spasm, irregular in character, with deviation of the head and face. The conjunctiva was insensitive. After about half a minute of this spasm, which was quite characteristic of epilepsy, it suddenly ceased; he gave a scream, harsh, but of volitional character, quite unlike the laryngeal cry of epilepsy, and then "co-ordinated" movements set in, struggling, throwing about of the arms and legs, quasi-volitional in character. Then his face, which had lost its initial distortion, broadened into a good-natured grin, and the attack was at an end. Observation of the attack left no doubt of its nature, which was confirmed by observation in hospital, where the patient had frequent simple epileptic fits consisting only of tonic spasm, but often with a severe fall. The case illustrates the diagnostic difficulties which arise, because the patient was unaware of his absolute loss of consciousness, and denied its occurrence, and the hysteroid stage was at one time prolonged. From the account of friends the attacks might have been regarded as purely hysterical, but they often occurred during sleep, which is a definite epileptic feature.

The recognition of an epileptic seizure preceding the hysteroid state is often very difficult if we have merely the friends' account of the compound attacks to guide us, and it is not always easy even when we can see an attack itself. When there is a severe epileptic convulsion, or even characteristic tonic spasm, as in the case just described, a hysteroid state succeeding it is clearly post-epileptic; but the slighter epileptic attacks may be difficult to discern. Their common features have, however, been already described. The most important indication is a sudden fall and a moment's stillness before the co-ordinated movements begin, and the fact that such attacks of *petit mal* occur also without the hysteroid sequel. Many such patients have had, in the past, severe epileptic fits. Long recurrence of attacks is also, as just stated, almost conclusive. So also is an aura common in epilepsy, especially one related to the special senses. It is remarkable how constantly a diagnosis made from these general characters is afterwards confirmed when the patient is admitted to the hospital and the attacks can be observed.

If a patient has epileptic fits succeeded by hysteroid convulsion, extreme caution is necessary in diagnosing the occurrence of independent hysteroid seizures, since the probability in such a case is great that these are really consecutive to slight epileptoid seizures. But in some cases the evidence of their independent nature is very strong, since they occur without any initial stage, resembling an attack of *petit mal*, and often occur under conditions of emotional excitement, and develop gradually.

(5) *Nature of Epileptoid Attacks.*—The last question in diagnosis is: If the convulsion is recognised to be epileptic in character, is it the manifestation of idiopathic epilepsy,

or is it due to reflex irritation, to toxæmia, or to organic brain disease, active or quiescent?

Reflex Convulsions.—Among the cases commonly regarded as the effect of reflex irritation are the convulsions which occur in young children at the time of the first dentition. In almost all such cases the general evidence of retarded development termed "rickets" may be recognised, and the convulsions are the result of the peculiar irritability of the nervous system which accompanies that constitutional state, and has been already discussed. The convulsions are usually referred to dental irritation. Such irritation may act as an exciting cause, but it is certain that the backward dentition is only part of a general retardation of development, the effect of which on the brain is to dispose to the occurrence of convulsions. Unless the nature of the cases is recognised and the diathetic state is treated, the convulsions may continue, and may persist as idiopathic epilepsy, or, after having occurred for two or three years, they may cease, to recur as epilepsy at the second dentition or at puberty. Moreover, it is especially common for such convulsions of rickets to persist in families in which there is an inherited tendency to epilepsy. Hence we can only make an absolute distinction from epilepsy while the evidences of rickets are still present. When these have passed away, and the convulsions persist, they are not separable from idiopathic epilepsy.

Convulsions at the period of the second dentition are very rare except in those who possess an inherited tendency to epilepsy, or have also suffered from convulsions in infancy, which have left a predisposition. Too often they continue after any dental irritation is over. The diagnosis, then, of simple reflex convulsions from dentition must always be made with hesitation; in most cases persistent fits are essentially epileptic.

Of other sources of reflex irritation which may cause convulsions, the most frequent are intestinal worms. They are of diagnostic importance chiefly in cases of recent fits, or in which only one convulsion has occurred.

The form of convulsion may sometimes lead to a suspicion that it is due to worms. It may be quite similar to the convulsions of idiopathic epilepsy, but frequently is slighter in degree, and consists only of tonic spasm. In the intervals between the fits convulsive starts are especially common.

In such cases, especially in children, this cause should be borne in mind, and excluded by careful inquiry and, in most cases, by the administration of vermifuge remedies. Tape-worms and round-worms are both occasional causes. The existence of the former may generally be ascertained without difficulty by the frequent passage of joints. It is doubtful whether threadworms cause fits, in spite of the great irritation of which the patient is conscious. When convulsions excited by intestinal worms have continued for several months, they usually do not cease when the exciting cause is removed. The repeated fits have apparently induced a state of the nervous system similar to that which exists in idiopathic epilepsy, from which these cases cannot then be separated.

Another occasional cause of reflex convulsions is the irritation of indigestible food. The fit is usually isolated, and occurs soon after the ingestion of the food, which is usually of such a character as to suggest itself at once as the cause. For instance, a medical student, whose bowels had been confined for a week, ate a hearty lunch of pickled mackerel, and drank with it a quantity of milk. He then had some beer, and went to a post-mortem examination, in the course of which he had a convulsive fit. There was no inherited tendency, and he had no fit before or afterwards.

The diagnosis that a fit which follows, and is no doubt excited by, injudicious food, is nothing more than a reflex convulsion, should be made with some caution, in children as well as in adults. It is doubtful whether such reflex convulsions occur, except in individuals who are specially predisposed. There may be an inherited or acquired tendency to epilepsy, and the fit thus excited may be simply the first of a long course of attacks. Again, the predisposition which permits the excitant to be effective may be commencing organic disease of the brain, which, at the time, has caused no other symptom. I have seen several instances of this. For instance, a little girl, apparently in perfect health, swallowed a piece of slate pencil, and had a fit a few hours later. The slate pencil was expelled, and at the end of a week, having had no other fit, she was apparently well. But a fortnight later she returned with headache, paralysis of some cranial nerves, and optic neuritis. She was thought to have tubercular meningitis, but the course of the symptoms was slow, and when she died, about two months later, a large glioma of the pons Varolii was found. Again, another child, apparently in good health, had a convulsion after some scarcely judicious food. The child seemed well afterwards, but a few days subsequently had another fit, then became febrile, and a fortnight later the signs of tubercular meningitis were distinct, and of this she died. In these patients, before the commencing disease in the brain caused other symptoms, it produced such an irritability of the nerve-centre that a slight cause led to a convulsive seizure. Thus the possibility that a fit, apparently excited by digestive derangement, may be the earliest expression of serious brain disease, or of the state of nervous system which causes epilepsy, must always be borne in mind.

In the very rare cases in which irritation of a cerebro-spinal nerve excites convulsions, the evidence of this is usually distinct, and the cases have been already sufficiently discussed (p. 29). In these also the possibility of cerebral disease must be remembered. The case has been mentioned of a man who, after injury to the left arm, had convulsions which began in the injured part. But in a few weeks he presented symptoms of a syphilitic tumour of the opposite hemisphere, and treatment for this permanently arrested the attacks.

Toxæmic Convulsions.—Of convulsions due to the presence in the blood of poisonous substances, only three forms are of importance in the diagnosis of epilepsy—those due to alcohol, to the blood-state in kidney disease, and to lead. All these may cause convulsions of epileptoid character and chronic course, which are independent of any actual visible lesion in the brain.

The cause of the attacks which are due to alcohol is usually sufficiently obvious. They usually recur after alcoholic excess. They are general convulsions, sometimes frequently repeated, which present nothing distinctive in their characters. The distinction from idiopathic epilepsy rests upon the ascertained relation of the attacks to their cause.

The convulsions which occur in acute uræmia rarely present diagnostic difficulty, since the other signs of this blood-state are usually sufficiently obtrusive. But it is important to remember that kidney disease may be attended by recurring fits, indistinguishable in their characters from idiopathic epilepsy. They are met with chiefly in cases of granular kidney. The diagnosis rests, of course, on the detection of the signs of renal disease. In most cases the pulse is incompressible and the heart hypertrophied, and, since the heart and urine should be carefully examined in every case of epilepsy, the condition ought never to escape detection. I have met with this renal epilepsy in a young man, but it is much more frequent in persons beyond middle life, in whom the possibility of its existence should always be suspected.

Chronic convulsions from lead poisoning may bear a close resemblance to the fits of idiopathic epilepsy. They are bilateral, and may begin with a special sense aura. Their diagnosis rests on the detection of their cause. In all cases of epilepsy in persons whose occupations expose them to the risk of lead poisoning, the gums should be carefully examined. Not unfrequently the convulsions are associated with other symptoms, such as wrist-drop, due to the same cause. It must not be assumed, however, that in every case of epileptic fits in the subjects of lead poisoning the convulsions are due to this cause. Epilepsy may have commenced in early life, before the patient began to work with lead. Moreover, the subjects of chronic lead poisoning often also suffer from chronic renal disease, and it may be doubtful to which cause the fits are due. The convulsions which are associated with severe cerebral symptoms due to acute lead poisoning are, of course, sufficiently distinctive.

It is important to remember that convulsions from lead and alcohol may persist after their original cause has ceased to operate, and, in such cases, the effect of that cause, and of the repeated convulsions, has apparently been to excite in the brain a pathological state similar to that which exists in simple epilepsy.

Convulsions from Organic Brain Disease.—The convulsions of idiopathic epilepsy have to be distinguished from those due to organic brain disease, active or stationary.

Convulsions often attend the onset of a sudden cerebral lesion, or occur during its early stage. They may occur, for instance, at the onset of cerebral hæmorrhage, or, more frequently, of cerebral softening from vascular occlusion, but the lesion which causes the convulsions causes also other obtrusive symptoms, hemiplegia, &c., which declare its existence. A convulsion beginning unilaterally may cause transient hemiplegia ("post-epileptic paralysis," p. 117); and if the convulsion is the first from which the patient has suffered, a doubt may exist as to whether both convulsion and paralysis are not the result of an acute cerebral lesion. The weakness succeeding the hemiplegia is usually slight, and is most marked in the limb (arm or leg) in which the fit commenced; it passes away in the course of a few hours. When, on the other hand, the convulsion and paralysis are the result of a cerebral lesion, the paralysis is usually at first complete; it affects both arm and leg, and it usually lasts for days or weeks. In most cases of post-epileptic paralysis, the patient has had other fits beginning unilaterally, with the same sequel. In cases of softening from vascular obstruction, convulsions may occur a few days or a week after the onset, apparently from irritation adjacent to the lesion. In these cases the convulsions succeed paralysis, and the recognition of their cause presents no difficulty.

Local active brain disease, especially tumour and local chronic meningitis, may cause convulsions closely resembling those of idiopathic epilepsy. They usually begin locally, and affect one side first or only. In all cases of convulsions so commencing, and also in the case of those beginning with a visual or auditory aura, the probability of this cause must be borne in mind, and careful search made for other symptoms of organic disease. The mere fact of local commencement and deliberate march does not, alone, constitute evidence that there is organic brain disease (such as tumour or meningitis), since fits occasionally begin thus in idiopathic epilepsy. Moreover, the convulsions of organic disease may not begin locally, but in the same manner as the fits of idiopathic epilepsy. In such a case, however, organic disease would not be thought of unless there were other symptoms to suggest it, whereas, if fits begin locally, this should be the first idea.

The other symptoms which commonly concur with convulsion from active disease are the following:—headache felt not only after the fits, but during the intervals; hemiplegic weakness, persisting; paralysis of cranial nerves; vomiting; optic neuritis. Syphilis is so frequent a cause of such organic brain disease as causes local convulsions, that a history of it gives significance to symptoms otherwise doubtful. The mere occurrence of convulsions in a subject of constitutional syphilis does not prove the existence of organic disease, since general fits may be due to idiopathic epilepsy, and the two diseases only accidentally coincident. But if the convulsions begin locally, and thus indicate some local change in the brain, a history of constitutional syphilis makes it extremely probable that there is organic disease of syphilitic nature.

Convulsions may not only result from active brain disease, but also from a stationary lesion of the brain which has become quiescent, or has even undergone cicatrisation, so that merely a scar may be found on post-mortem examination. They may result from a mere contusion of the cortex from injury, or from old disease, such as a tubercular or syphilitic process. The other symptoms of active disease, headache, optic neuritis, paralysis, may pass away, but the convulsions may persist by a continuance of the morbid state of the grey matter which was damaged by the irritation of the lesion in its active stage. For instance, a man who had constitutional syphilis suffered from convulsions, optic neuritis, and severe headache, and a syphilitic tumour of the brain was diagnosed. The optic neuritis and headache passed away, but the fits persisted, and some time afterwards he died in a series of severe convulsions, killed apparently by their violence. Post mortem there was found the atrophied remains of a gumma in the lower part of the right frontal lobe. In a large number of these cases the convulsions begin locally, and are thus of the character to suggest organic disease. Whether they begin thus or not, the history of the case shows that at the onset there were symptoms of organic disease, and these make its nature clear. Frequently also some of the early symptoms persist, as some hemiplegic weakness, or optic-nerve atrophy such as is produced by neuritis.

In a very important group of cases an acute lesion of the brain—in most softening from thrombosis or embolism—causes at first hemiplegia and afterwards convulsions, which may persist as long as the individual lives, although hemiplegia passes away wholly or in part. These cases have been described at length. In many cases the disease dates from childhood, and then seems often to be due to local polio-encephalitis, or sometimes to a thrombosis in a surface vein. In most the history of the onset will indicate the nature of the case. Whenever epilepsy begins with severe convulsions in early life, especially if these occurred during the course of, or after, an acute specific disease, during any period of general prostration, or after a blow or fall on the head, inquiry should be made for hemiplegia after the early fits. The parents frequently omit to mention it, attaching chief importance to the convulsions. Sometimes, indeed, they fail to recognise it, especially in a young infant. In most cases the recurring convulsions affect first or only the side on which the hemiplegia existed, and they frequently begin locally.

Such an organic cause may always be suspected if the initial fits were unilateral and severe. Sometimes a cortical lesion in early life is only followed by fits at the epileptogenic period of puberty. The causal relations may then be obscure, but such attacks have still the feature of local commencement. It is important to note that minor attacks may be associated which are quite like those of idiopathic epilepsy, but they seldom develop until convulsions have occurred for some years.

Local commencement is, however, sometimes met with in attacks of idiopathic epilepsy. One symptom, no doubt, indicates local instability, but of a nature different from that of organic disease. In such cases there is no history of severe convulsions at the onset or in early life, still less of hemiplegia; there is commonly a family history suggestive of inheritance, and the local commencement may vary as regards its side.

A special sense aura is rare in organic epilepsy, and if it involves more than one of the special senses, or is psychical in nature, the case is almost certainly idiopathic.

General Paralysis of the Insane.—In some cases of general paralysis, recurring convulsions, often unilateral, and sometimes varying in seat, constitute the first symptom of the disease, and the affection may thus in the early stage be mistaken for epilepsy. In most cases other symptoms of the disease are present, tremor of lips or tongue, defective articulation, or a tendency to optimism. If these symptoms are present in a case of short duration, in an adult man, the disease is probably general paralysis and not epilepsy. A history of syphilis is usually to be ascertained, but it may involve the difficulty of distinction from an organic lesion of the brain, just described. The difficulty is sometimes considerable, since a convulsion with slight subsequent weakness may be due to either cause. As a rule the nature of the case is cleared by a short period of observation.

Simulated Convulsion.—Epileptic fits may be simulated; they are chiefly shammed by professional beggars* and by soldiers. Commonly the pretended attack is so unlike a true epileptic fit that its nature is at once recognised. Approximate simulation is only possible by a person thoroughly familiar with the characters of the disease. The symptoms may then leave a skilled observer in doubt, especially when cyanosis is induced by voluntary fixation of the chest, and a piece of soap in the mouth facilitates the frothing at the lips. The artificial convulsion may bear some resemblance to the aberrant forms of genuine epileptic fit with extensor rigidity of limbs and fine tremor-like clonic spasm; but the abnormal features usually enable the nature of the case to be suspected. Circumstantial evidence, the occurrence or repetition of attacks under conditions in which they may be advantageous to the individual, often, however, first affords confirmation of the fraud.*

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* The simulation of epilepsy is a matter of careful training among professional beggars and criminals, to whom it is useful, not only as a means of obtaining alms, but also for escaping penal labour. An interesting account of the confessions of one of these individuals, who succeeded for several years in deceiving English police and prison surgeons, and was ultimately detected in America, has been given by Dr. C. F. Macdonald in the 'American Journal of Insanity' for July, 1880.

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* Many years ago the late Sir Russell Reynolds and I were walking in the street when a man fell in front of us in an apparent epileptic fit. Neither could say whether it was real or simulated. The latter is probable, although the choice of spectators may have been unintentional.

CHAPTER XI.

PROGNOSIS.

THE prognosis in epilepsy involves several separate questions: (1) the danger to life; (2) the prospect of a spontaneous termination of the disease; (3) the prospect that by treatment the disease may be (*a*) cured, or (*b*) the attacks arrested.

(1) *The danger to life* in epilepsy is not great. Alarming as is the aspect of a severe epileptic fit—imminent as the danger to life appears when the patient is lying senseless with livid, swollen, and distorted features, and looking "as if strangled by the bow of an invisible executioner,"* it is extremely rare for a patient to die during a fit. The apparent danger to life is from the arrest of breathing and interference with the action of the heart by the tonic spasm, which sets the frame in stillness. But the asphyxial state of the blood induces remission in the spasm, by which its effect is gradually lessened, and even patients with grave heart disease pass through the most severe fits with apparent impunity.

The chief danger to life in an attack is the liability to "accident," that is to some secondary effect by which death may be caused. One of these is common "choking." If an attack occurs during a meal, food may get into the air-passages; vomiting after an attack may have the same result. Hence there is more danger to patients who vomit while unconscious after a fit than to others; the tendency to this is frequent in a few cases, but quite absent in others—indeed, in the majority. Another danger is due to the tendency of the patient, after an attack in bed, to turn on to the face, and be suffocated in the post-epileptic insensibility, another feature that is special to some cases only.

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* Radcliffe.

There is also some risk of death by other forms of accident to which the attacks expose the patient. Epileptics whose fits occur without warning, are sometimes injured by falling on to a fire, and the burns thus received may be serious and dangerous. But the commonest mode of accidental death in epilepsy is by drowning. The fit not only occasions the fall into the water, but effectually prevents any effort to escape. Hence epileptics are sometimes drowned in a very small depth of water, as in a ditch, or a bath.

Yet the danger of such accidental death is unquestionably small. The proportion of cases in which it occurs is not ascertainable, but, if all cases of epilepsy are considered, it is minute. Death from the direct severity of a fit is also very rare. But an important exception is presented by the cases which pass into what is termed the "status epilepticus" (described at p. 207), in which severe attacks recur very frequently, recovery from one being imperfect before another comes on. This state is one of considerable danger: it is, however, rare, and the liability to it cannot be regarded as measurably increasing the risk of death in consequence of the disease.

(2) *What is the prospect of a spontaneous cessation of the fits?*—The tendency of the disease is to self-perpetuation: each attack facilitates the occurrence of another, by increasing the instability of the nerve elements. Hence the spontaneous cessation of the disease is an event too rare to be reasonably anticipated in any given case. Occasionally, however, convulsive attacks in infancy, which continue after all cause to which they could be attributed has passed away, cease spontaneously at four or five years of age; sometimes they do not afterwards recur, but too often they come on again at the period of the second dentition or at puberty. Attacks which have commenced before, and continued until puberty, rarely cease at that period. The expectation, firmly

rooted in the popular mind, that attacks in girls before the onset of menstruation will cease when this has occurred, is unjustified by facts. It is a time at which there is a special tendency for the malady to commence, and least disposition for it to cease. After twenty years of age spontaneous cessation does sometimes occur, and it becomes more frequent as life advances. It is, I believe, a more frequent event than writers on the disease have usually admitted, but is not sufficiently common to be reckoned on as a practical element in prognosis.

Marriage, as a rule, makes no difference to epileptics. The attacks occur afterwards with the same frequency and severity as before. In some patients they cease during pregnancy, but they usually return when the pregnancy is over.

(3) *The probability of cure or arrest by treatment.*—The probability of a spontaneous arrest of the disease being so small, the next question becomes of paramount importance: What is the prospect of cure of the disease, or of arrest of the fits by treatment? Of all questions in the prognosis of disease it is the most difficult to answer, and no other is, perhaps, put with the same anxiety and frequency.

The facts relating to the cure of the disease are extremely meagre. Few cases can be watched sufficiently long to enable a cure to be confidently affirmed. Moreover the cases in which the best results are obtained, in which no more fits occur, are precisely those that are never heard of again. Such cases are, I am sure, far more numerous than is imagined. The few which are incidentally heard of at a subsequent time make it certain that they represent a much larger number of whom no trace is obtained.

As far as is known, the only method of cure is by securing arrest of the fits for a considerable time. Hence the practical question is, what is the prospect, in any given case, that an arrest of the fits can be obtained by treatment? In order to ascertain how far the various conditions of the disease can be taken as indications of the amenability of a case to treatment, I have compared the facts of a series of cases, in which no good was done by any method of treatment, with another in which the attacks were arrested by treatment as long as the patients remained under observation, all cases known to have relapsed being excluded. The conclusions agree with those that have been impressed on me by the observation of individual cases.

The facts which make arrest more likely have a similar significance regarding the probability of the more frequent result of treatment, a great diminution in the attacks without complete arrest. This often transforms the life of a sufferer from the disease. At the same time, only absolute arrest of all attacks places the patient definitely on the road to an ultimate cure.

The points examined with reference to prognosis are—(1) sex; (2) age at commencement; (3) duration; (4) inherited tendency; (5) exciting cause; (6) frequency of attack; (7) condition of patient under which attacks occur; (8) existence of an aura; (9) mental state; (10) character of the fits.

Sex.—In the cases which were unimproved the females preponderated (as in the whole of the cases of epilepsy analysed, see p. 2). In the cases in which the attacks ceased under treatment the sexes were affected equally. Hence the males were relatively more numerous, and the females less numerous, among these cases, than among the entire series, and it may therefore be concluded that the prognosis is slightly better in males than in females.

Age.—The proportion of the cases commencing under twenty in which arrest was obtained is considerably less than the proportion of cases commencing over twenty, the difference amounting to about 13 per cent. The period during the first twenty years of life at which the disease commences has little influence, but the prognosis seems somewhat better in the cases which commence under ten than in those which commence between ten and twenty; arrest is more frequently obtained. The cases which commence in women at the second climacteric, also obstinate, seldom yield to treatment.

Duration.—As all writers on the subject have stated, the prognosis is favourable in inverse proportion to the duration of the disease. The prognosis is by far the most favourable in cases which have lasted less than a year. Having regard only to the probabilities that treatment will arrest the fits, or will have no effect whatever upon them, it appears that if the disease has lasted less than five years the probability is a little in favour of arrest, but that if the disease has existed for more than five years it is more likely that no effect will be produced than that complete arrest will be obtained. Of course, at any age the probability of amelioration, of a diminution of the fits, it may be to one fiftieth or one hundredth of their former number, is always greater than either of the two eventualities which are now, for the sake of bringing out more clearly the prognostic indications, alone under consideration.

Hereditary predisposition might reasonably be expected to have an unfavourable influence on prognosis, to render it less likely that an arrest of the fits would be obtained. As Herpin first pointed out,* such is not the case. From observation of individual cases I have been strongly impressed by the frequency with which heredity exists in the cases in which treatment has the most marked effect, and the fact is clearly brought out by a comparison of cases. The explanation may be that a slighter exciting influence is effective in such predisposed cases, and is more easily counteracted. It does not of course follow that a permanent cure is more readily effected in such cases. The greater readiness with which the fits are arrested may be counterbalanced by a greater tendency to relapse. A similar fact has been observed with regard to insanity; heredity does not lessen the probability of recovery from a given attack.

Illustrations of this fact are often met with, such as was strikingly presented by the following case:

A young man, whose mother's two sisters and one brother were epileptic, had his first fit at nineteen, and soon afterwards came under treatment. The attacks were at once arrested, and they remained absent for two years. Then, at twenty-one, they recurred, and, having had two fits in one week, he came back to the hospital. Each fit was preceded for an hour or two by a "nasty feeling of numbness in the tongue," but there was no immediate warning. In the fit consciousness was lost, and his tongue bitten. A scruple of bromide twice a day was ordered, and he had no other fit, and after two years' treatment he was discharged. The fits remained absent for a year, and then he had some more, and again came to the hospital. The attacks at once ceased under the same treatment, but he had occasional "sensations" in the tongue, which became more frequent when the same dose of bromide of sodium was substituted for bromide of potassium, but on the resumption of the latter with Indian hemp, although only once a day, the sensations ceased, and he had when last heard of had no symptom for a year.

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* Loc. cit., p. 516.

An equally striking illustration was presented by the patient, mentioned on a previous page, fourteen of whose relations suffered from epilepsy. The attacks commenced at ten years of age, and she was thirty-seven when she came under treatment. The interval, before the treatment commenced, was two weeks. The attacks occurred during sleep only, and never during pregnancy. They were severe epileptic fits, with tongue-biting. Under treatment the attacks practically ceased. As long as she was taking ℈j or ʒss of bromide at bedtime she had no fit, but after she had been a year, eighteen months, or two years without an attack, she would discontinue the bromide and presently have another fit. This went on for seven years, and the bromide was just as effectual as when she first came under treatment.

Exciting Cause.—The recognition of an exciting cause for the disease, *i. e.* a cause for the first fit, has little influence on the prognosis. Such a cause can be traced in about the same proportion of the cases which are arrested, and of those which are not influenced by treatment. No indication can be drawn from the character of the exciting cause in the cases in which one can be traced.

Frequency of Attack.—The prospect of a complete arrest of the fits is extremely small if severe attacks occur daily. It is greatest if the attacks occur at a longer interval than one month. There is not, however, a simple relation between length of interval and prognosis, because attacks which occur at the regular interval of a month are less frequently arrested than those which occur at a shorter but irregular interval.

The Condition in which the attacks occur, whether during the waking or sleeping state, or both, is also of significance. In the cases of arrest the attacks occurred in only one state in three times as many cases as in both sleeping and waking. In the cases without improvement a larger number had attacks in both states than in one only. It is clear, therefore, as far as these figures go, that the prognosis is far better if the attacks occur in one state only than if they occur in both. If occurrences during the sleeping or the waking state are compared, the prospect of arrest is found to be rather better when attacks occur only during sleep.

Aura.—The occurrence of a distinct aura seems to render the prognosis a little more favourable, but the difference is not sufficient to possess much significance. The form of aura seems to have little influence on prognosis.

Mental Change.—The existence of considerable mental change renders the prognosis unfavourable. Mere slight loss of memory is, however, of little significance. It may pass away if the attacks are checked by treatment.

Character of Fits.—Regarding the influence on prognosis of the character of the fit, it is to be remarked that the major attacks are influenced by treatment much more readily than the minor fits. The former often cease before the latter are materially lessened, and it is not infrequent for the minor attacks to persist in spite of treatment, although the former are completely arrested. Sometimes indeed the slight attacks become more frequent, and they are then often difficult to influence.

To sum up the prognostic indications in idiopathic epilepsy:—The prognosis is slightly more favourable if the patient is of the female sex, and distinctly more favourable if the disease begins over twenty than if it commences between ten and twenty. It is better the shorter the duration of the disease, and when the disease is inherited than when no heredity can be traced. It is better the longer the interval between the severe fits, and is least so in the cases in which attacks occur almost daily. It is better if the attacks occur in the sleeping or in the waking state only than if they occur in both. It is better if there is no considerable mental change, and if the attacks are all of the severe variety, than if there are minor seizures, and better if the attacks are preceded by an aura than if they occur without warning.

It must be recognised, however, that a really good prognosis can seldom be given unless all attacks are averted by bromide in moderate doses, such as fifty or sixty grains a day. Larger doses may diminish the fits but seldom effect a cure, and this is true also of the substitutes for bromide. Successful results are sometimes obtained, but they are few. The prognosis in cases which "cannot bear" bromide is generally unfavourable so far as the prospect of a cure is concerned. But in all cases the ultimate result largely depends on the ability to secure persistence with effective treatment for a sufficient time, two or three years. Less does not secure the stability that allows the controlling agent to be discontinued with impunity. It is the case whatever the agent is. Premature cessation of treatment is certain to involve recurrence, and the fresh start is harder than the first. There is no short road to a cure, and the prognosis must be largely influenced by the presence of the necessary patience and wisdom.

Organic Epilepsy.—In the cases in which the attacks are due to an old stationary organic lesion of the cortex the prognosis is much less favourable than in the idiopathic cases. It is influenced by duration of the disease and interval between the attacks, just as in the idiopathic form, but, as might be expected, the effect of remedies is less marked, and this variety furnishes some of the most obstinate cases which come under treatment.

Post-epileptic Symptoms.—The prognosis in cases with post-epileptic hysteroid convulsion is, on the whole, the same as that of the minor epilepsy which commonly precedes them. The hysteroid sequel often ceases under treatment, while the minor attacks continue. On the other hand, post-epileptic automatic action, whether quiet or violent, commonly continues if the fits which excite it persist. This is especially true of the violence which merits the name of epileptic mania, and the attacks of which it is the sequel are also especially difficult to arrest.

NOTE.

THE MORTALITY FROM EPILEPSY.

The actual mortality from epilepsy itself is very difficult to ascertain. The present Registrar to the National Hospital for the Paralysed and Epileptic, Dr. Farquhar Buzzard, has, however, ascertained the number and causes of death in the cases of epilepsy in the hospital during the 34½ years in which records have been kept. The number of cases is 2828. The number of deaths is 38, but no details of the immediate cause are to be found in 4. Of the rest, 15 died from some chronic unrelated disease existing before admission, and 7 of some acute disease contracted after admission. Only 12 deaths were directly connected with the disease. One was certified as asthenia, one as asphyxia (an instance of turning over after an unnoticed fit in the night), 7 were due to the status epilepticus, and 2 to intercurrent mania and exhaustion.

CHAPTER XII.

TREATMENT.

WITH the exception of those diseases for which nothing can be done, the treatment of few is so difficult as that of epilepsy. It is easy to understand the source of the difficulty. We have seen that epilepsy is a self-perpetuating disease. The renewal of the constituents of the nerve elements, after their release in functional action, occurs in such a way as to facilitate the subsequent recurrence of the same functional activity. (See p. 224.) This residual effect is beyond our power of prevention; it is a condition of nerve action, essential to normal function, and inevitable in that which is morbid. It is perhaps more surprising that epilepsy should be often arrested, and sometimes actually cured, than that it should so often resist our efforts to control it. That we can exert some influence on the disease is due to the fact that, by the presence of certain chemical agents, the morbid process may be to some degree controlled. By maintaining the control, a considerable degree of stability seems to be induced by a corresponding influence on the process of nutrition, in consequence of which the transient influence of the agent employed becomes fixed, and it can at last be discontinued.

CAUSAL TREATMENT.

The direct treatment of all diseases is subordinate to that which can be achieved by the removal of their causes, when this is possible. The first question should always be, Can the attacks be ascribed to any cause which can be removed?

Unfortunately, it is seldom that the search for such a cause of epilepsy is successful. Occasionally a source of peripheral irritation may be found, capable of exciting what are called "reflex convulsions." Intestinal worms are the most frequent; their possibility should be borne in mind in all recent cases. Round-worms and tapeworms are those chiefly effective. Great as is the local irritation caused by thread-worms, they seem hardly ever to excite convulsions. When there are any indications of the presence of the more effective varieties, there should always be a renewed recourse to vermifuge agents. Unfortunately, when fits due to them have recurred many times, they often do not cease on the expulsion of the exciting agent. This has been already mentioned (p. 29).

Indigestible food is frequently regarded as the cause of the first fit, and sometimes with reason; but it is effective chiefly in predisposed subjects, in whom no subsequent care of diet suffices to secure persistent freedom. This cause is so common that the special result can only be explained by a predisposition, in consequence of which recurrence of the fits is almost constant. In the very rare cases in which a cause of peripheral irritation of a cerebro-spinal nerve exists, especially when the fits begin in the irritated part, the removal of the source of irritation is important—such as the excision of a scar involving a nerve, or, if that is impossible, resection of the nerve going to the part.

When alcoholic excess has preceded the onset of attacks, and especially if it distinctly excites them, abstinence is of the utmost importance, and more often suffices to arrest the disease than any other causal measure. In the rarer cases in which chronic lead-poisoning can be found, the arrest and treatment of this are necessarily essential; but the attacks may persist although freedom from the toxic agent is secured. Pronounced anæmia is not often met with as a cause, but if it exists, it should be dealt with by appropriate treatment. The existence of epilepsy need not interfere with the administration of iron.

MEDICINAL TREATMENT.—BROMIDE.

Apart from the removal of any discernible causes of the disease just considered, and apart from general regimen and surgical measures, to be mentioned later, the treatment of epilepsy is purely medicinal. It has always been so, but in a far more pronounced degree since the almost accidental discovery, more than forty years ago, of the influence on the disease exerted by the combinations of bromine. Their effect many times exceeds that of all other agents put together. The influence of chemical agents on that which is a process of vital action may seem anomalous, until we remember that all activity in the body seems to consist in chemical processes occurring under the influence of life. This is true of the release of energy by means of muscles, and it is so with the liberation of what we call "nerve energy." There seems no doubt that the process depends on the release of atoms from the complex molecules to unite more closely with those of oxygen brought to them by the plasma from the blood, and thus release the force which kept them apart in the nerve-substance. As we have seen in the account of the pathology of the disease, this must be regarded as a vital chemical process.

The history of the use of bromide in epilepsy has not been described, as far as I have been able to find. Its use seems to have spread quickly, but there is a distinct sequence in published observation. Bromide of potassium was introduced into the 'Brit. Pharmacopœia' in 1835, on account of its supposed value in enlargement of the spleen. It was removed in 1851 as useless. In 1857 ('Lancet,' i, 528) Sir Charles Locock advocated its use in epilepsy occurring at the menstrual periods. The first testimony to its service in ordinary epilepsy (combined, however, with iodide) seems to have been given by Sir Samuel Wilks in 1861 ('Med. Times,' Dec. 21st). A year later ('Lancet,' Dec. 10th, 1862) Dr. Goddard Rogers published some cases successfully treated at the West London Hospital by bromide of ammonium, which Dr. Duncan Gibb had been using there for some time to lessen the sensibility of the throat. Dr. Ramskill, early in 1863 ('Med. Times,' i, 221), published a single case treated by bromide of potassium at the Queen Square Hospital, and it seems to have been constantly used there through that year by himself, Dr. Radcliffe, and Dr. Hughlings Jackson (see note on the subject, 'Med. Times,' Feb. 13th, 1864, p. 173). In

February, 1864 ('Dublin Med. Journal') Dr. McDonnell advocated its use in epilepsy, and he has been associated with Locock as deserving the credit of its employment, but he had evidently been anticipated. After this date records become abundant. No earlier observations seem to have been made abroad.

Although the effect of bromide is far from certain, and variable in degree, it frequently causes an arrest of the attacks, which, if sufficiently maintained, may result in an actual cure of the disease, that is, the ability to leave off the agent without recurrence of the fits. The proportion of cases in which this result is achieved is no doubt small, but it cannot be estimated, and is certainly greater than is demonstrable. Many patients who come to a hospital and are treated with bromide for a few months, then cease attendance; it is certain that some of these never have another fit, because in the case of a few the fact is learned accidentally at some subsequent period. It is the same with patients who are seen in private. Those who are seen again and again are those whose attacks continue; but many, after visits for a year or two, having had no fits, are seen no more. They go on with the medicine for a time, but without doubt many would come again if the attacks recurred. The significance of the fact that they do not return, is confirmed by some who are afterwards heard of as having been permanently free after discontinuance of the medicine. It is true that they are few, but when the cases are considered which are found, after several years, to have remained free, the conclusion is irresistible that a much larger number are actually cured than can be discerned. The conditions of the problem involve sources of fallacy against which simple statistical methods are unable to guard. The proportion in which a cure results is no doubt small compared with the larger number of those who suffer.

In another small proportion of cases bromide is found to be without any influence for good. Between these two classes come the vast majority of cases, in which the attacks are reduced in frequency, and often in severity, but are not arrested. The reduction is often considerable. Instead of two or three fits a week, a patient has a fit only every one or two months. Instead of one fit a month, he may have only one fit a year. But the result often means the ability to pursue an occupation which would be otherwise impossible. It means the transformation of home life from one of constant dread, to one of comparative confidence and tranquillity. The difference to a family if a patient has a fit only in three or four months, instead of an attack every one or two days, is very great. Hence it is that many patients who are not cured, nevertheless cannot do without bromide; and the number of the epileptic out-patients attending at the National Hospital for the Paralysed and Epileptic, who have been doing so for many years, is very great. The strongest testimony to the influence of bromide on epilepsy is afforded by the amount used there. For certainly thirty years the physicians have been constantly endeavouring to discover an efficient substitute for it, with little success, and the amount used at the hospital during the year 1899 was almost two tons. Most of this large amount was given to out-patients regularly attending on account of the benefit they derived. It was given in response to a need which it alone could supply. It is a striking testimony to the good that is done even when a cure is not achieved. In therapeutics, relief is only second to a cure.

Certain as is the fact that bromide lessens the tendency to spontaneous discharges, long as we have been familiar with that influence, we are still without knowledge of the precise manner in which it acts. It is certainly in consequence of the presence of bromine in the haloid salt, for a corresponding combination of chlorine has almost no influence. Bromide is said to cause contraction of the small vessels; but even if it has this effect, it is not likely that its influence on epilepsy is thus produced. It must be conveyed in the plasma directly to the nerve structures of the brain. In some way it restrains the undue tendency to the escape of the atoms of the nerve-substance to combine with the oxygen of the plasma, but we do not know whether it does so by the mere effect of its presence, or by yielding its bromine to combine with the constituents of the nerve element. We have no ground for accepting one view rather than another. We know, however, that the effect of its presence is transient, that its absence is quickly followed by a return of the tendency to convulsion. If bromine is detached and enters into the constitution of the nerve-substance, it must be a feeble union, since its effects are so short-lived. On the whole, it seems possible that the effects are produced by undecomposed bromine, perhaps passing into the nerve structures, but restraining in some way the morbid affinity for oxygen, although not yielding its bromide to form part of the molecules of the nerve-substance, as arsenic seems to do in its action on the nerves.

Whatever be the mechanism, the influence of bromide on the functional action of the cerebral cortex admits of no doubt. Experiment testifies to it clearly. Under normal conditions electrical stimulation of the motor cortex gives rise to convulsion. If the stimulation is gentle, the spasm is localised to the part whose centre is excited; if more energetic, the spasmodic movement spreads, and may become a general convulsive attack. If bromide has been previously given, and in proportion to its amount, this effect is lessened. A slight stimulus may have no effect; a strong one may have no more result than a slight one; and at last a toxic point is reached, at which an excitation has no effect which would normally cause a severe convulsion.

These facts enable us to understand the influence of bromide on epilepsy, since the disease, as far as we can discern, depends on an abnormal readiness for action in the cortex of the brain, in consequence of which the process of union of atoms of the nerve-substance with those of adjacent oxygen occurs without the normal stimulus which should be needful to liberate them from their complex combination, and release the energy previously held latent. This seemingly unexcited action we call "discharge." Bromide lessens the tendency to this, as it does the excitability of the cortex. But how the affect is produced we are not yet able to discern.

The agent must, however, be conveyed by the blood equally to all parts of the nervous system; but its influence has been proved experimentally to be much greater on the central organs than on the peripheral nerves. Its action on the motor cortex of the brain has been conclusively demonstrated, but it is not limited to the motor region. Large doses, such as two drachms or more, cause some headache, mental dulness, slight impairment of articulation, and diminished sensitiveness, especially of the pharynx. Still larger doses (200 grains) are said to lessen reflex action in the spinal cord, and even cutaneous sensibility. That the effect is due to an action on the centre is shown by the fact that it is the same in a limb from which the blood-supply has been cut off. Voluntary processes are little impaired. Bromide antagonises strychnia, apparently by neutralising the effect of the alkaloid on the motor centres. Its influence seems to be the result of an action on the nerve elements themselves.

The excretion of bromide by the urine begins in fifteen minutes after it has been taken, and continues, gradually diminishing, for three weeks, if the dose has been large. Repeated doses are to some extent cumulative, according to the amount which has been given, and many effects of a single large dose are observed after repeated smaller doses.

The peculiar character of the action of bromide on the brain is presented by its influence on sleep. It does not compel sleep, as does opium, but seems to permit, or rather promote it, by lessening mental activity and dulling the perception of external influences.

Combinations.—Several combinations of bromine with a base have been used in epilepsy. Those which have been chiefly employed are bromides of potassium, sodium, ammonium, lithium, and strontium. Bromide of zinc has been also used, but can be given only in small doses, so that its influence depends on the zinc rather than on the bromine. Opinions have differed much regarding the value of the several compounds. The differences in effect are conceivably due to two causes: first, the influence of the base; and secondly, the proportion of bromine contained. On account of the former, some compounds, especially that of potassium, have been thought to be particularly "lowering." It is difficult to perceive in practice the indications of such an influence. No greater "lowering" influence can be discerned from bromide of potassium than from bromide of ammonium.

This suggests a doubt whether the bromide is decomposed in the system, or whether the base exerts definite influence. It has been thought that any other bromide than that of sodium is decomposed by the chloride of sodium in the blood, and bromide of sodium is necessarily produced. This is apparent ground for attaching weight to the character of the base, but the fact is not certain. We are able to discern almost nothing of the chemical changes which occur in the blood.* Still, the impression that the base has a special influence, has led to the extensive adoption of the practice of giving a combination of two or three bromides, so that no alkali shall preponderate. I have never been able to perceive any practicable objection to it, or any foundation for its supposed advantage.

The second distinction between the bromides seems to possess more weight. The amount of bromine in each differs considerably; and since the presence of bromine in the salt is unquestionably the essential element in its action, it is reasonable to expect the influence of a compound to be proportioned to the amount of bromine contained. This in the compounds most in use is as follows:

Lithium bromide	92	per cent.	bromine.
Ammonium † ,,	81½	,,	,,
Sodium † ,,	77½	,,	,,
Potassium ,,	67	,,	,,
Strontium ‡ ,,	47½	,,	,,

Thus the bromide of lithium should be the most effective, and that of sodium less useful. But observation affords little or no confirmation of the opinion. Strange as it is, sometimes one, sometimes another salt, seems to answer better in a given patient, without relation to the nature of the base or the amount of bromine it holds. Difficult as this may be to understand, it receives strong testimony from the use of the several bromides at the Queen Square Hospital. For certainly thirty-five years there has been a free choice for the prescribers, among the three salts in most use. (The bromides of lithium and of strontium have been used chiefly during the last seven years.) If any one combination had possessed distinct advantages, these could not have escaped discernment, considering that the practice of the hospital includes careful observation on in-patients, as well as the more numerous, and perhaps equally useful, opportunities of observation on out-patients. The amount of each bromide used has therefore great significance. The following are the quantities for the year 1899:

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* I have been at considerable pains to ascertain what can be said, with authority, regarding the changes bromide undergoes in the blood, and how far the modern doctrine of "ions" can be adopted. The result is negative. We know nothing of "ions" except in simple solutions, and the chemical condition of simple saline solutions in the blood is unknown, at least in so far as to justify the inference of ionic changes.

† This is the percentage in the anhydrous sodic bromide of the B.P. The crystallised salt contains two atoms of water, and therefore only 57 per cent. of bromine.

‡ The crystallised bromide of strontium of commerce contains six atoms of water.

Bromide of potassium	3664 lbs.
" sodium	313 "
" ammonium	114 "
" strontium	6 "
" lithium	3 "

The amount of bromide of potassium is about twelve times as much as that of sodium, and nearly ten times as much as all the other bromides taken together, including that of sodium The potassium salt was that which first came into use, but the others soon had ample trial, and the proportion of the several salts now employed conveys strong evidence that no other salt has much superiority.*

Administration.—In the treatment of epilepsy, whether with bromide or any other agent, the endeavour must be to find a dose (or combination with other drugs to be presently mentioned) which arrests all attacks, slight and severe, and which the patient can bear. This must be continued persistently, without intermission, until it is reasonable to suppose that stability has been established, and then should be discontinued by gradual diminution. Hence it is important that the dose should not be larger than can be continuously borne, no larger than suffices to arrest the attacks and provide a margin of safety against accidental exciting causes.

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* Some years ago I carefully compared the relative effect of bromide of sodium and of potassium in fifty cases. In many no difference was perceptible; but in at least half the cases the sodium bromide seemed less useful, dose for dose. But the conclusion deserves little weight, because the sodic salt was probably crystallised and hydrated, and therefore contained much less bromine.

Unfortunately it is seldom that this persistent continuity can be secured. In most cases, when the patient has been free from fits for a few months the medicine is discontinued. The daily dose is an annoyance, or friends are ready with the assurance that it will "do harm to go on with so depressing a medicine;" and, strangely, often the same advice is given by some medical practitioner. It is left off, and in a week or two the patient has another fit. Often the discontinuance is due to the prevalent idea among practitioners that the administration of bromide is incompatible with the treatment of any intercurrent disease, even a trifling cold. On the least excuse, its omission is insisted on. Many relapses are thus produced. The intermission is, indeed, justifiable in acute diseases with considerable fever. It is very rare for an attack to occur during the pyrexia, but as soon as this ceases the tendency returns. In many slighter ailments, the medicine is omitted when the utmost that is justified is a reduction in the dose, and perhaps a simplication in the combination in which it is given.

There is no doubt that, but for these causes, the number of definite cures of epilepsy would be larger than it is. The cases in which attacks are absolutely arrested are on the road to a cure, and it is in these that the temptation to omit medicine is greatest. Patients are advised to leave it off, from fear of injurious consequences, for the most part illusory. Any harm which bromide does passes away soon after the administration ceases, and no permanent injury to the nervous system ever results. But to its influence is ascribed all that occurs during its administration. It has undesirable effects in some cases, which will be considered immediately, and the conditions which involve most liability to them. All that is here asserted is the fact that unfounded apprehension often lessens the prospect of a cure, and more frequent prospect of great improvement. It is surprising how widespread is the prejudice against the use of bromide in the only effective way—continuous administration. It is less surprising how often gratitude results when the prejudice is overcome. There are, indeed, many cases in which effective doses cannot be borne; some for which it is unsuited; and the discernment of the cases in which other agents are more useful should always be an object of continuous effort.

It is important to note that the influence of bromide is cumulative. Attacks may not be at once arrested, but may cease gradually in the course of two or three months' administration. But for the continuous administration of bromide, the minimum dose that is effective should be employed, on account of the length of time for which it must be given. The large doses, advocated by some Continental physicians, cannot be borne for long in the majority of cases. The best results are obtained by from 60 to 80 grains a day, in two or three doses. Often, indeed, two doses of 25 grains are enough, and are generally well borne. The cases in which there is permanent arrest of the attacks seldom need more than 60 grains a day. If 30 grains three times a day do not produce arrest, larger doses are seldom successful. Bromide is better given after meals, because it need not be suddenly introduced into the blood, and on account of the advantage of combining some arsenic to prevent acne. If two doses are given, one may be after breakfast and the other at bedtime. If attacks occur only in the night, a single dose of 30 or 40 grains at bedtime is often successful. Attacks which occur soon after rising in the morning are sometimes not arrested by the night dose, and it may be well to give an early morning dose, without arsenic, on first waking; or, if with arsenic, after a cup of tea. In the case of children, the dose should be relatively smaller than for adults, but not in proportion to the years. It is useless to give less than 5 grains to a child one or two years old; and afterwards the dose in grains which is given twice a day may be double the years of the child's age. Of course in all cases the frequency of attacks and the time at which they occur has to be taken into consideration.

For the reasons already stated, it is difficult to ascertain the cases which are actually cured, in which the patient remains permanently free after the discontinuance of the treatment. The following examples, however, seem conclusive.

A barrister, aged 35, with strong heredity, had his first fit at thirty. At thirty-five he had the second known fit, very severe, during sleep. He took 30 grains of bromide every night, but after eighteen months reduced it to 20 grains, and at the end of the second year he had another, after a period of work on hasty and insufficient meals. The dose was increased to 30 grains for two years, and then reduced to 20, which he continued for several years. Indeed, he was reluctant to leave it off. But he has now had eighteen years of entire freedom, doing all the time the extremely hard work of a most successful barrister.

A Scotch solicitor, aged 24, with no heredity, had his first attack at seven, and they continued at intervals of a few weeks until he was thirteen, and then ceased until twenty-two. They then recurred, and continued at intervals of a month or two. The convulsion was general, and the attacks occurred always when awake. On 25 grains of bromide with 1½ of bromide of zinc twice a day he had no other fit. After two years he took it only once a day, and after another year gradually diminished it, and left it off. By inquiry I have ascertained that he has now taken no medicine for seven years, but has continued absolutely free, although he has got married, and has been doing the work of a busy solicitor.

In the following case the attacks were of a minor character only, that which is commonly most obstinate.

A girl of 10, with heredity, had many minor attacks every day. She was unconscious for a moment, and stopped whatever she was doing, and then went on with it as before. On 10 grains of bromide of potassium three times a day she had still ten of these daily; but an increase to 15 grains caused a gradual diminution at the end of the first month. At the end of three months they ceased entirely. After six months more treatment she ceased attendance at the Hospital, but I have ascertained that she has kept perfectly free from fits, although it is now fourteen years since the medicine was discontinued.

These cases should dispel any doubt of the occasional cure of the disease. I am convinced that they might be multiplied many times were it possible to ascertain the after-history of the large number of patients who are entirely free from fits as long as they remain under observation, often in spite of gradual [illegible] the dose. Page after page might be oc [illegible] ch cases. A remarkable fact is [illegible] ch cases present an inherited tendency to the disease, an illustration of the fact, already mentioned, that heredity does not make the prognosis less favourable.

A lad of 18, whose mother was epileptic, had both severe and slight fits, the latter consisting of the warning of the major attacks, with brief unconsciousness. On 25 grains of bromide and 3 of lactate of zinc all attacks ceased, and he remained free for three years. He then died of an acute disease.

A girl aged 27, whose brother suffered from fits, had her first attack at nine, immediately after a fright. She had had them ever since, and when she came under treatment they occurred at intervals of about six weeks. Each was preceded by a feeling of "lostness" for a few seconds, then her arms became stiff, and she lost consciousness, and the fits were followed by heavy sleep. The sensation sometimes occurred without a fit. A scruple of bromide was given three times a day. She had no other fit. For twelve months she had occasionally a "faint feeling," and at the end of that time the bromide was reduced to two doses a day. The "faint feelings" became less and less frequent, and at the end of the second year the bromide was omitted altogether, and quinine was given for a few months. Five years after the cessation of treatment, and seven after the last fit, there had been no recurrence. She had married and borne children.

A girl of 13 had suffered from severe epileptic fits, at night only, for six months, the interval being about three weeks. In addition she had minor attacks in the day, about one a week. Fifteen grains of bromide and 1 of bromide of zinc were given twice a day. The severe fits ceased, but she had one or two minor attacks during the first three months. These also ceased when the zinc was increased to 2 grains, and when last seen she had been four years without any attack.

A girl of 28 presented an illustration of a morbid tendency, which cannot be described as heredity, but is remarkable as apparently due to the consanguinity of parents. The father and mother were first cousins. Of the children, three boys and two girls were congenitally blind, and one boy was epileptic as well as the patient. The fits were not frequent, but ceased entirely for the two and a half years she was under observation.

The arrest of fits is, of course, not equivalent to the cure of the disease. In only too many cases, when the attacks have been arrested for a long time by treatment, they recur when that treatment is discontinued. In some cases the patient remains free from fits for one, two, or three years, and then the attacks recur. But, in the majority of cases which relapse, recurrence takes place within a few months of the cessation of treatment; and if, after such cessation, the patient has passed a year without an attack, there is good ground for the hope that they will not return, an expectation which is strengthened by every subsequent year of freedom.

If a relapse occurs, the fits are less readily arrested by the same treatment than in the first instance. A very frequent occurrence is that a patient, after a few months' freedom from fits, discontinues the medicine and relapses. He comes again; the fits are again stopped, but less readily than before; again he omits the treatment and the attacks recur, and are then not completely arrested. Hence it is most important to maintain by long treatment the initial arrest of the fits. The longer the period since an attack, the greater, *cæteris paribus*, is the probability that the arrest will continue after the medicine is discontinued. Hence treatment should be continued, without any reduction in dose, for two years after the last fit, or even longer, the period varying according to the duration of the disease and frequency of attacks before treatment was commenced, and the readiness with which cessation is obtained. The dose should then be gradually reduced during a third year, and only then be entirely discontinued. It should never be suddenly left off. Most cases of the *status epilepticus* are due to such sudden discontinuance, under some vague impression that it is doing harm.

Treatment must be persisted in as long as minor attacks continue. The occurrence of these makes it certain that the patient will have a severe fit soon after treatment is stopped, or is much reduced. These remarks apply to all agents, as well as to bromide.

The recurrence of fits which have been arrested by treatment may be spontaneous, or it may occur only on a considerable exciting cause. For instance, in the following case an accident reproduced attacks which appeared to have been effectually arrested.

Fanny P—, whose father's brother was insane, had her first fit at ten, while unconscious after a fall out of a "boat-swing." She came under treatment at the age of fourteen, having then a fit every fortnight. Under treatment (which was continued for three years) the attacks ceased entirely. She then took no medicine, and had no attack for five years. At the end of that time she was thrown out of a dogcart, again struck her head, and had a fit a few minutes afterwards. They recurred every ten days, until she came under treatment nine months later, when they again ceased.

If a relapse occurs after bromide treatment has been discontinued, the drug should be continued the second time for a much longer period than the first, as in the following case, which illustrates also a relapse after a long period of freedom.

A man, whose father's mother was insane, had his first fit, without known cause, at seventeen, and came under treatment at the age of twenty-two. Bromide (℈j bis) at once arrested the fits, and after he had been under treatment for nine months, having no fit, he ceased attendance, and remained free from fits for four years. He then had another attack after running quickly up to the top of a factory. Bromide was again given, and he had passed a year and three quarters without an attack at the last note.

When bromide 'cures' epilepsy, removing altogether the state of the nervous system to which attacks are due, this effect must be ascribed to a permanent nutritional change of the same character as the effect which is produced by its presence in the system. This effect seems soon to cease, but if this condition on which it depends is maintained for a long time, nutritional changes occur by which the effect is maintained after the bromide is withdrawn. In the hope of producing this alteration more readily and in greater degree, I have tried the effect of giving very large doses of bromide for a comparatively short time. The method was to begin with doses of two or three drachms of bromide every second or third morning, and to increase the dose to four drachms every fourth morning, and six drachms or an ounce every fifth morning. These large doses were given after breakfast, in a tumblerful of water. If the salt is not well diluted they cause epigastric pain and vomiting. As a rule, when the dose reaches half an ounce, some drowsiness and mental dulness follow during the rest of the day, but are gone the following day. Some patients complain only of headache, a dull constricting sensation, which is relieved by the application of cold. The susceptibility of different patients to these large doses varies exceedingly. Some patients cannot bear more than four drachms; in others a dose of an ounce produces little change from their normal state. These doses usually lower the frequency of the pulse 8-14 beats, but I have been surprised to observe no appreciable difference in the temperature or in the reflex actions.

The maximum dose was reached in two or three weeks, and repeated three or four times, and the doses then gradually reduced, so that the whole course lasted six or seven weeks. It is to be remembered that the object of this method was to attempt to further the *cure* of the disease. It was therefore only adopted in cases in which the attacks were influenced in a marked degree by bromide. If the bromide was omitted at the end of the six or seven weeks' course, the patient as a rule remained free from fits for a longer time than would follow, in similar cases, so short a period of treatment by bromide in ordinary doses. The attack, however, afterwards recurred if all treatment was relinquished, and bromide had to be resumed in ordinary doses. The results obtained were, on the whole, disappointing, since in no case was the course followed by freedom unless bromide was continued. This maximum-dose course seems to exert some influence, but it ceases after a few months.*

OTHER SALTS OF BROMINE.

Bromide of Strontium has been used and praised during the last few years, especially because it is thought to have less tendency to cause acne. This seems to be in harmony with the small amount of bromine contained in proportion to the base,† and so also is the large dose which has been considered necessary by those who have most strongly recommended it, amounting to two or three drachms a day. It is not, therefore, surprising that some observers (as Vallon) have given it up, because with doses which could be borne, the attacks were more frequent. My own experience has shown no superiority over the other bromides. It should also be noted that, when given to animals in large doses, Féré found its poisonous influence was five times as great as that of bromide of potassium.

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* It has been referred to as if a lasting effect was claimed for it. On the contrary, I only advocated it as probably of some value as an initial stage in persistent treatment.

† The commercial bromide of strontium contains six atoms of water of crystallisation.

Bromide of Nickel has been tried, but can only be given in a minute dose, which is without influence. The same may be said of the combined *Bromide of Rubidium and Ammonium*, the dose of which is 2 to 5 grains. *Bromide of Zinc* depends for its activity on the metal, under which it will be mentioned.

Bromide of Camphor, which has been praised by Bournevilles, can only be given in a dose of a few grains. This is insufficient to enable the bromine to have any influence, and the camphor seems to act only as a nervine stimulant. I have seen no benefit in any case in which I have tried it.

Bromalin is a name given to a compound of which the full designation is bromethylformin. It has been given on the doubtful theory that the inconveniences of bromide are due to derangement of the intestinal tract, and it is supposed to be decomposed in the stomach into bromine and formaldehyde, which is an antiseptic. It contains only half the amount of bromine as bromide of potassium. Its great expense is a hindrance to its use, and it has been found to have little influence on the attacks, and readily to cause acne.

Bromipin is a combination (prepared by Merck) of bromine and sesame oil (a grain-oil obtained from India, used for the lubrication of machinery). The compound presents the first apparent opportunity of giving pure bromine, so far as ingestion is concerned. The same compound is prepared in England by Martindale and by Morson, under the more appropriate name of *Brominol* or *Brominoleum*. It is made with 10 per cent. of bromine, or with 33 per cent. The latter contains half the quantity of bromine as bromide of potassium; a drachm of brominol (bromipin) corresponds to half a drachm of potassium bromide. It has been extensively praised on the Continent, as causing no rash and none of the depression which sometimes results from bromide, and as being very effective in its influence on the attacks. The advantages are also claimed for it that it can be given by rectal injection, or rubbed into the skin. When given by the mouth, it can be only absorbed when the oil is acted on in the intestine by the pancreatic secretion, the bile, etc. The combination with the oil is very close. According to my own experience it has much less tendency to cause acne, but also much less tendency to arrest the attacks. On comparison with an equivalent amount of bromide of potassium. the difference in its influence is considerable. Bromine can be detected in the urine after its administration. The compound can be given either as an emulsion, with a few drops of chloroform and mucilage, or mixed well with an equal volume of syrup, or in capsules. The pure oil is not unpleasant. The compound deserves further trial, but it seems doubtful whether it will be found of much value.*

Hydrobromic Acid cannot be given in sufficient quantity to secure an influence on the attacks. Moreover, it cannot exist in the blood in its acid state, and must be at once converted into bromide of sodium.† The acid is occasionally useful, when it is desirable to acidulate a mixture containing bromide, as, for instance, if strychnia is also given. Hydrobromate of strychnia is readily soluble. Caution should be observed in giving strychnia with unacidulated bromide.‡

Many other combinations of bromine have been tried without result. The addition of iodide has seemed useless in cases in which there was no syphilitic lesion of the brain. For pure epilepsy an alkaline salt of iodine is as useless as one of chlorine.

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* To prevent misconception it may be well to state that the results were chiefly obtained with Merck's bromipin, but no difference could be discerned between it and the English preparation. The comparatively slight effect suggested that much might pass through the bowel unabsorbed, but some analyses of fæces of patients taking three drachms daily (33 per cent. preparation) gave no support to this view. It seems possible that the bromine passes into the blood in some organic combination which lessens its influence.

† In case it should be desired to give bromide of potassium without the knowledge of the patient, it may be well to state that 20 grains of Pot. brom. result from the combination of 17 grains of Pot. bicarb. with 140 minims of Acid. hydrobrom. dil.

‡ A pharmaceutical chemist received a prescription for bromide and strychnia, without any acid, but in which the water was sufficient to make a perfectly soluble mixture. Going to the seaside, he got a local chemist to dispense the mixture double strength. Probably an unduly alkaline bromide was used. He did not notice a precipitate which formed in the mixture, and apparently took the whole of the strychnia in the last dose; in consequence he almost died of strychnine poisoning. The original prescription (not given by myself) was apparently safe, but the incident is instructive.

DISADVANTAGES OF BROMIDE.

Two classes of unpleasant symptoms may result from bromide,—mental and physical depression, and the bromide rash. The former is due to an amount greater than the individual can well tolerate; the latter is due to cutaneous idiosyncrasy.

Cerebral Depression.—The general brain depression which is produced after some time by large doses is not surprising. Although the influence of bromide seems primarily and chiefly to be exerted on the motor centres of the cortex, many of its physiological effects show that, as might be expected, its influence is exerted widely, and it is not surprising, therefore, that other functions of the brain should, after a time, be interfered with. The effect, when extreme, is mental as well as motor lethargy, loss of memory, lessened sensibility (especially of the pharynx), and even dribbling of saliva. Such effects are only met with in most cases in which a dose of 100 or 120 grains a day has been given for some time, or with smaller doses in patients with originally weak brains. Sometimes the effect is simply mental depression and slight lethargy. Very rarely is there any definite derangement of the mental functions.

It is important, moreover, to note that these slighter symptoms are often met with when comparatively small doses of bromide arrest attacks. They have also been met with in cases in which attacks cease spontaneously from some undiscerned cause. They are thus apparently often due to the cessation of the habitual discharges to which the brain is accustomed, for which nerve energy is generated, which, undischarged, seems to act widely on the brain and depress its functions. If a patient, whose attacks have been arrested by bromide, passes into such a condition, another attack at once clears off the cloud which seemed to be upon the brain. The improvement occurs irrespective of the continuance of the bromide, showing clearly that it is the effect on the brain of the cessation of the discharges, and not the agent which produces this, which causes the condition. The discharges may be compared to the [illegible] necessarily produced. [illegible] general increase of pressure, which [illegible] attributed to the [illegible] are sometimes the effect of the [illegible] the inevitable result of the process by which [illegible]

[illegible]

[illegible]

of suppuration small; sometimes the area of suppuration is much larger than corresponds to the base. Almost as frequent, however, and more so at the commencement of the rash, are small pustules, with little redness, and papules which do not always reach the stage of pustules, and are often hard. I have known this form of rash to be mistaken for varioloid. Large pustules are occasionally seen in which there are many white points of cheesy or purulent material, and occasionally there are large and superficial bleb-like collections of pus surrounded by a little redness. Guttmann has shown that the presence of bromine may readily be demonstrated in the contents of the pustules. The larger pustules cause much irritation, and are often painful; they may leave permanent scars, so that, if numerous, considerable disfigurement on the face results, from the pitting and from the reddening and thickening of the skin between the pustules.

This troublesome symptom may be almost entirely prevented by the addition of arsenic to the bromide. It was recommended by Echeverria in 1870.* From three to five minims of liq. arsenicalis added to each scruple of bromide will usually remove the eruption, if moderate, and greatly diminish it, if considerable. The amount of arsenic necessary, however, depends on the individual tendency to the rash. When the eruption has thus been removed, if the arsenic is omitted, the bromide being continued, the eruption returns. (See, for examples, 1st ed., p. 263.) Since the influence of arsenic has been known, the terrible faces, so common before from this cause, are no longer seen.

But the use of arsenic for this purpose is not without disadvantages. It hardly ever causes the multiple neuritis, which has been so common when arsenic has been taken in beer, nor does it cause herpes zoster. The difference may be due to the fact that bromide lessens metabolism in the nervous system. But it occasionally causes, at least apparently, slight gastro-intestinal irritation, and trifling conjunctival irritation. The most serious effect is the pigmentation of the skin, which is seen in perhaps a fifth or sixth of the cases to which it has been regularly given for several years. It usually begins by minute spots of brownish pigmentation, often following the spots of redness. They may coalesce and cover large areas, but usually leave a few unpigmented white spots of skin. Sometimes the pigmentation seems diffuse from the first. It is most marked on the neck, and parts exposed to pressure, and is sometimes mistaken for Addison's disease. It often causes concern, but we have no other means of preventing the acne, and the choice has sometimes to be given—which is preferred. The borrowing is always chosen.

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* Loc. cit., p. 318.

I have known a peculiar form of trophic change in the hands to result, due to neuritis, but have never met with typical peripheral neuritis from the administration of arsenic with bromide.

ADJUNCTS TO BROMIDE.

Those drugs which are of definite service when given alone will be mentioned later. Some agents, especially of organic nature, of little or no value alone, seem often to augment the influence of bromide. In a considerable number of patients the effect of the several combinations was carefully tested by giving, first, bromide alone for several months; and, when its influence was ascertained, the second drug was added to the same dose of bromide, and the effect compared.

Digitalis is a very old remedy for epilepsy. It was recommended by Parkinson in 1640[*], and has been, perhaps for as long a time, a popular remedy for the disease in the West of England. Given alone, its influence is slight. It may lessen the frequency of attacks, and sometimes even arrest them for a short time; but I have not met with any case in which, under its use, frequent attacks remained absent for more than a month or two. It is, however, often distinctly useful in addition to bromide. Attacks which continued on bromide only, ceased entirely on bromide and digitalis.*

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[*] See Parkinson, 'The Theater of Plants' (1640), p. 654, "Foxglove" (Vertues):—"It hath beene of later experience found also to be effectuall against the Falling-sicknesse, that divers have beene cured thereby; for after the taking of the decoction of two handfulls thereof, with foure ounces of Pollipody, of the oake bruised made in Ale, they that have been troubled with that disease 26 yeares, and have fallen once in a weeke or two or three times in a moneth, have not fallen once in 14 or 15 moneths, that is until the writing hereof, which I thinke may be said to be an absolute cure, not to be presumed that after so long stay it should returne againe."

Among the cases which are most strikingly benefited by the combination are those which are complicated by cardiac disease, or dilatation, irregularity, or undue frequency. The use of these agents in such cases is an obvious measure. The only cardiac condition in which I have known it to have a prejudicial effect on the attacks is in aortic regurgitation accompanied by considerable hypertrophy, a condition in which the use of the drug is to a large extent contra-indicated for cardiac reasons. It seems especially to augment the influence of bromide in nocturnal epilepsy.

By what influence digitalis is useful is not known. It steadies the circulation and increases the tone of the small arteries, and thus, doubtless, renders equable the supply of blood to the brain. But this effect is produced through the nervous system; that on the heart is supposed to be exerted through the vagus, an indication of a central influence. Moreover, large doses of digitalis, if continued for some time, cause symptoms of cerebral and spinal disturbance, giddiness, amblyopia, dilatation of the pupil, and even diminished reflex excitability in the spinal cord. These are commonly explained as secondary to the disturbance of the circulation, and the accumulation of carbonic acid in the blood, but it is more likely that they are due to a direct influence upon the nerve-centres.

The greater effect of the combination of bromide and digitalis than of either given alone is clearly shown by the following case:

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* These paragraphs are almost unaltered from the first edition (1880). The value of these cardiac tonics (digitalis, adonis vernalis, convallaria, strophanthus) has been recently advocated by Bechterew ('Neur. Cent.,' 1898), who especially esteems convallaria. Hence it is curious to read in Parkinson's 'Herbal' (loc. cit., 1640, p. 1407), under "Tillia, the Lime or Linden tree."—"The flowers of the Lime tree, and of Lilly Convally, being destilled together, the water of them is much commended against the falling-sicknesse; the destilled water of the barke is of the same effect."

In a girl, aged 20, without heredity, one to three fits had occurred weekly since sixteen years of age, distinctly epileptic, with tongue-biting and without warning. Her pulse was 96, irregular in force but not in frequency; the impulse slapping, but in the normal situation. On tinct. digitalis ♏v *ter die*, she had one fit a fortnight; on pot. brom. gr. xx *bis die*, she had a fit each week; on a combination of the two twice daily, she had not a single fit for three months, and then ceased attendance.

The following cases also illustrate the beneficial influence of the combination:

A girl, aged 21, had two fits at six months old, and no others till thirteen, when they recommenced, and continued once a month, chiefly at catamenial periods. They were severe, epileptic in character, occurring both when awake and when asleep, and when awake there was an epigastric aura. There was a mitral murmur and dilatation. After two years' treatment by bromide, alone and combined with iron, and also with arsenic, she was having several fits every month. There was some improvement with bromide and belladonna, but after some months' treatment three fits still occurred each month. Tr. digitalis ♏v was then added to the same dose of bromide (℈j ter die), and she had not a single severe fit for six months, although "sensations" continued a few times a week. She then ceased attendance, but almost immediately had another attack, and they recurred on the same treatment about one a month, and then almost ceased. During the next two years she had only one attack every four or six months, but if the medicine was discontinued the attacks immediately recurred.

A man, with some dilatation of the heart and irregularity, began, at 44, to suffer from "twitchings in the face and hands," which continued, until at 46½ he suffered from nocturnal fits, epileptic in character, general, but more severe on the right side. At 47, when he came under treatment, he had two a week. On bromide only they ceased for three months, and then recurred, and continued in spite of ʒss of bromide three times a day. During three months' treatment on bromide (℈j) and belladonna (♏v), three times a day, he had a fit each fortnight. The same quantity of tincture of digitalis was then substituted for the belladonna, and the fits ceased for six months, except on one occasion when he omitted the medicine for a fortnight. At the end of six months the dose was lessened, and he had some more attacks, but they again ceased when the dose was increased, and he remained free for several months, and then had two more.

A boy, aged 12, had suffered from fits since he was sixteen months old, being then backward in teething, in walking, and in talking. His legs retained traces of old rickety curvature. The attacks occurred during both waking and sleeping states, at intervals of a week. When awake they were preceded by a visual aura, "a ball of fire before the eyes," always red, and seeming to get nearer and nearer until he lost consciousness. The convulsion commenced in the right side of the face, and was more severe in the right limbs. Amm. brom. gr. xv, with Tinct. belladonnæ ♏v, first twice and then three times day, increased the interval to one month; but after six months' treatment the attacks still occurred. Tinct. digitalis, ♏v, was then substituted for the belladonna, and he had no fit for eleven months, when the dose was reduced and a fit occurred; but on again increasing the dose seven months passed, and then he had two fits, then an interval of two months, and on again substituting belladonna for the digitalis the attacks became more frequent.

A girl, aged 18, without heredity, came under treatment for fits which had occurred frequently since ten, and also at seven and two. The interval was about two days. The fits were without warning, and evidently epileptic. Bromide alone, and with belladonna, lessened a little the frequency of the attacks, but made no considerable difference, from twelve to sixteen fits occurring every month. Digitalis was then substituted for the belladonna, with the effect of reducing the fits first to four and then to two in the month, and then she passed four months without a single fit. At the end of that time they recurred, but again ceased for four months under the same treatment.*

Strophanthus.—The effect of strophanthus is sufficiently similar to that of digitalis to justify the expectation that it is equally useful as an adjunct to bromide. This is confirmed by observation, but I have not had the same opportunities for testing its influence on a large scale, free from distinct sources of fallacy.

Belladonna, like digitalis, rarely useful alone, sometimes distinctly increases the effect of bromide. It also has long been used, having been recommended by Mardorf in 1691, and at the end of the last century by Münch, Stoll, and Hufeland. I have met with no case of true epilepsy in which the attacks ceased entirely on belladonna, although, in several patients, the fits were reduced in frequency. As an adjunct to bromide, its utility is often unquestionable. It has been said to be most useful in cases of *petit mal*, but it is also effective in cases in which the attacks are severe, whether they occur by day or by night.

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* Many other cases were given in the first edition; but the evidence scarcely needs their repetition, or the narration of cases of similar significance since observed.

Its influence is probably due to its direct action on the central nervous system, which is, by large doses, first stimulated and then depressed. The effect is apparently similar upon the cerebral hemispheres, the spinal cord, and the vagus and cardiac nerves. Cerebral excitement is followed by coma; reflex action is at first increased and afterwards lessened; the heart is at first slowed, and afterwards accelerated.

The following cases illustrate the influence of the combination of belladonna and bromide in epilepsy. In the first three cases the bromide was given alone in the first instance, and the superiority of the combination was evident.

Emma R—, married, without heredity, came under treatment at 35, on account of fits which had occurred frequently since the age of 15. There was no warning, and consciousness was lost. Urine was often passed during the attack, and it was followed by some delirious excitement with erotic tendency. Several fits occurred every week. On Potass. brom., ℈j *ter die*, she had one fit the first month, none the second, and two the third. Tinct. belladonnæ, ♏v, was then added. She had one fit in each of the next three months, and then the attacks ceased. Four months later she had one. The treatment was continued for six months more, the dose being gradually diminished, and then she ceased to attend, and remained free from fits without treatment for twelve months. A recurrence then took place, but again ceased on the same treatment.

A woman, two months after a confinement, at 43, began to suffer from fits which recurred at intervals of a fortnight when she came under treatment at 49. The fits were epileptic, the warning being pain in the head. On bromide gr. xv. three times a day the frequency remained the same. The same dose of bromide with tr. belladonnæ ♏v, increased the interval first to three months, then to six months, and then to a year.

Many other cases could be narrated in which the result of the combination of bromide and belladonna was most satisfactory; but they do not afford conclusive evidence of the influence of the belladonna, since the combination was given from the first. (See 1st ed., p. 270.)

Atropine has been frequently employed in doses of one or two minims of the liq. atropiæ of the British Pharmacopœia, containing $\frac{1}{120}$ and $\frac{1}{60}$ of a grain of atropine. In some cases it seems to be more efficacious than the belladonna.

A boy, aged 16, whose father's mother was epileptic, had suffered for a year from fits, chiefly in the early morning, without warning, and distinctly epileptic. Two or three attacks occurred each week. On bromide (♏j *bis*) there was no attack for twelve months; then they returned, and he had an attack each month in spite of the bromide being given three times a day with ♏v of tinct. belladonnæ. One minim of liq. atropiæ was then substituted for the belladonna, and he had only two fits in five months. The dose of liq. atropiæ was then increased to two minims, and he had no fit for eight months. Then a slight attack occurred, but he afterwards had fifteen months' freedom on the same treatment.

Stramonium, another solanaceous plant, was recommended in the last century by Baron Störck. I have had no experience of its use; the commendation by some old writers is shared by such a vast number of herbs as to excite little confidence.

Cannabis indica, which was first recommended in epilepsy by Dr Reynolds,* is sometimes, though not very frequently, useful. It is of small value as an adjunct to bromide, except for the relief of headaches, when this complication is troublesome. But it is sometimes of considerable service given separately. It may be noted that the action of Indian hemp presents many points of resemblance to that of belladonna; it is capable of causing also delirium and sleep, first depression, and then acceleration of the heart, and also dilates the pupil. The cerebral excitement is relatively more marked, and the effect on the heart and pupil much less than in the case of belladonna.

In the following case its effect was far more decided than that of bromide. The case is remarkable, but I have not succeeded in obtaining a similar result.

John K—, aged 40, had suffered from fits for twenty-five years. They occurred during both sleeping and waking, at intervals of a fortnight. There was a brief warning, vertigo, then loss of consciousness, and tonic and clonic spasm followed by some automatism;—'acts strangely and cannot dress himself.' The attacks ceased for a time on bromide, but recurred when he discontinued attendance. He came again two years later; scruple doses of bromide of potassium three times a day had now no effect, and the fits, at the end of four months' treatment, were as frequent as ever. Ext. cannabis indicæ gr. $\frac{1}{6}$, three times a day, was then ordered; the fits ceased at once, 'a wonderful change' the patient declared. He had no fit for six months, and then, having discontinued attendance, the fits recurred, but were at once arrested by the same dose of Indian hemp. He continued free from fits for some months, until, during my absence, bromide was substituted for the Indian hemp; the fits immediately recurred, and he left off treatment. He returned to the hospital in six months' time, and on Indian hemp passed two months without an attack. In the third month another fit occurred, and the patient again ceased to attend, and did not return.

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* Loc. cit., p. 321. It has been also recommended by Sinkler ('Philadelphia Med. Times,' Sept., 1878).

Gelsemium.—The effect of gelsemium on the spinal cord, the respiratory centre, and the cranial nerves, suggested its trial. The results show that its value, alone or in combination with bromide, is not considerable, although now and then it seems to be of slight service.*

Hyoscine.—The sedative influence of the hydrobromate of hyoscine on the cortex has led me to use it in many cases of epilepsy in combination with bromide, but in only a few cases has slight benefit been perceptible. It is a drug whose action is cumulative, and it is not safe to give it by the mouth in larger doses than $\frac{1}{400}$ or $\frac{1}{300}$ grain. The most convenient form is in a solution of 1 in 1000.

OPIUM.

Opium was recommended in epilepsy by Paracelsus and by Crato and Quercetanus in the sixteenth century, and De Haen published a case in which large doses of laudanum, taken on the occurrence of the warning of a fit, cut short the attacks and cured the disease, a sequence of doubtful credibility. It was tried thirty years ago at the Queen Square Hospital, but without definite good. It has, for the most part, given place to other remedies. Its alkaloid, morphia, was also recommended and tried in a considerable number of cases, but without evidence of clear utility.

It is necessary to mention a method of treatment which was advocated, in 1893, by the great cerebral anatomist, Flechsig, and has received attention corresponding to his anatomical reputation. It consists in a six weeks' course of opium, in doses increasing to 15 grains a day. The opium is suddenly stopped, and bromide substituted, 130 grains a day for two months, then gradually diminished to 30 grains a day. The opium is regarded as a preparation for the bromide treatment. The period of opium treatment is one for some concern, and, indeed, Flechsig recommends that the patient should be watched throughout the course as one suffering from an acute disease.

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* Case quoted p. 272, 1st ed.

The verdict of those who have tried the method is almost uniformly unfavourable.* During the period of opium treatment, the attacks are seldom lessened, and patients have indeed been known to pass into the "status epilepticus." It is doubtful whether the subsequent administration of bromide in such large doses has more influence than it would have without the preceding course of opium. The benefit sometimes seen in old obstinate cases of epilepsy may be explained by the large doses of bromide administered after a period of freedom from its influence.

In connection with the use of opium, it should be mentioned that morphia is an agent which can only be employed hypodermically in epileptics with extreme caution. If an attack occurs, and the post-epileptic coma coincides with the sleep induced by morphia, the patient's life may be in great danger. For instance, an epileptic, after a surgical operation, was injected with a quarter of a grain of morphia to relieve great pain. As the injection was beginning to take effect, he had an epileptic fit, and immediately passed into a condition of profound coma, with infrequent breathing, and it was necessary to maintain artificial respiration for an hour. In another case, the combined effect of a fit and the same injection given just before it occurred, was such profound coma that the patient died; but it is uncertain whether the treatment for opium poisoning was adopted in this case.

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* Among those who have reported unfavourably are Donath, Bohme, Lannois Luske, Kothe, Homen, Ziehen, Warda, Bratz. The last, out of forty-six cases subjected to the treatment, met with three cases of the *status epilepticus*, and several cases of severe mental depression, on the substitution of opium for bromide previously given.

BORAX.

In inveterate cases of epilepsy in which bromide has no effect, borax may sometimes be given with considerable benefit.* In many of the cases, indeed, little effect is produced on the disease, but in some its influence is decided, and may be greater than that of any other remedy. The dose varies from fifteen grains to half a drachm, three times a day, although smaller doses may be used in combination with bromide. It is not well to begin with a larger dose than 15 grains, because, in some patients, it causes at first some diarrhœa, occasionally with dysenteric evacuations. This, however, which is not very common, disappears if the dose is reduced, and does not recur when the dose is subsequently increased. It has been pushed to 120 grains a day for a time, being afterwards reduced. One observer found that attacks were lessened in two thirds of the cases, and were arrested in a tenth. But it must be remembered that no comparison with bromide was suggested, except in old-standing cases in which bromide had failed.† It may be taken for a long time without inconvenience, but in rare cases an eruption of psoriasis has occurred—in one patient after it had been taken daily for two years, and in another after nine months' administration, and in the third after it had been taken for eight months. In each case, however, the eruption quickly disappeared when arsenic was added to the borax.

Robert C—, without heredity, began to suffer at 16 from fits. He came under my care at 37, when he was having the attacks frequently under bromide treatment, two having occurred during the preceding ten days. Before a fit, for three or four minutes, he was unable to speak or to write, although he could go on with his work. Then his head became confused, and he fell unconscious, with a variable amount of convulsion. Gr. xv of borax was ordered twice a day, and he had not a single fit for nine months, when he had another, and during the next twelve months he had a very slight fit every two or three months. In September, 1880, he omitted the medicine, and had two attacks. He then took borax continuously for two years, the daily dose being gradually increased to ʒjss, and then only had an attack at an interval of a few months. He said that borax had done him far more good than any other medicine,

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* The use of borax in cases which resist bromide was first suggested by me in the Goulstonian Lectures for 1879 (which formed the basis of this book). The opinions there expressed have been since repeatedly confirmed.

† See my Goulstonian Lectures, 'Lancet,' 1879, vol. i, and the 1st ed. of this book, 1880, p. 280. An account of careful observations confirming my conclusions was given by Risien Russell and Taylor, 'Lancet,' vol. i, 1890; but the number of writers who have described their experience to the same effect is very large.

The influence of borax over fits is very well shown in the following case, and it is interesting because bromide was more effectual after the borax treatment than before, and it shows clearly that if borax does good for a time, and then seems to lose its power, bromide should again be tried, even though it had little effect before.

Emma B—, single, aged 14, without heredity, had suffered from fits, evidently epileptic, for fifteen months. The attacks occurred during the waking state, at intervals of two weeks. They were without warning, and severe. Bromide alone was first given for two months, but the attacks were rather worse than better, several occurring each month. On belladonna alone the weekly average was 1 and 1·5. Ten grains of borax three times a day was then given, and the fits at once ceased, and she had no attack for five months. The dose was then ordered to be taken only twice a day, and she had a fit almost immediately. The third dose was resumed; a fit occurred the following month, and then four months passed without an attack. A fit occurred in each of the two following months, so bromide (℈j three times a day) was substituted, and for fourteen months she had no other fit.

Borax may be given in combination with bromide, and deserves a trial in obstinate cases; but the dose must be smaller, 5 to 10 grains, and the results are, on the whole, less encouraging.

The following is a curious instance of the effect of combination with gelsemium:

Arthur O—, without heredity, suffered from fits in infancy, between one and two years of age; dentition and walking were both late. The attacks continued from that time until he came under treatment at 17. The interval varied from one to six days. The fits were sudden, without warning, slight, followed by some automatism. During two months' treatment on bromide the attacks were uninfluenced. On five grains of oxide of zinc three times a day he had two fits a month, but only one fit a month on seven, ten, and twelve grains. Tinct. ferri perchlor. was then substituted; during the first month he had one fit; during the next month none, but afterwards the attacks became more frequent, and on other treatment the attacks became very numerous, so that in one week he had fifty-four. Ten grains of borax three times a day were then given, and for the first week he had no fit; then some attacks in the night, but none in the day; the latter recurred after some weeks, and the dose was increased to twelve grains, and five minims of tinct. gelsemii added. The fits at once stopped, and when last seen he had not had a single fit for sixteen months.

NITROGLYCERINE.

The general opinion is in agreement with that of Osler, that nitroglycerine exerts no appreciable influence on the attacks of epilepsy. A case presently to be narrated shows, however, that this is not always the case, and that there are some cases in which it is of singular service. Moreover it seems often to be a useful adjunct to bromide in cases with feeble circulation, coldness and pallor of the fingers, and small, slow pulse. Such patients seem to derive benefit from it, although it is not possible to obtain satisfactory evidence of an influence on the attacks. It is certainly also useful in the cases in which periodical headaches persist with epilepsy, instead of ceasing, as is the common rule. It is best given in combination with bromide as the liq. trinitrini of the Pharmacopœia, a one per cent. solution of nitroglycerine in alcohol. For the stability of the nitroglycerine, however, it is necessary to make the mixture acid, which is readily done by the addition of ten or fifteen minims of dilute hydrobromic acid to each dose. This solution is a convenient method of giving it in combination for any purpose.

The following is the case referred to as an instance that nitroglycerine may be useful in ordinary epilepsy. The conditions of the patient remained precisely the same during the whole course of treatment.

A girl of 16 had her first fit at 13. They rapidly increased in frequency until, when she came under treatment, they occurred every night, severe, with frequent biting of the tongue. She was also liable to migrainous headaches. On half a drachm of bromide of potassium each night she had thirteen in a week. With belladonna added, she had eighteen, twenty-four, and twenty-eight in successive weeks. The bromide was then stopped, and $\frac{1}{100}$ grain of nitro-glycerine was given three times a day. In the first week she had eight fits, and in the second three. The dose was increased to $\frac{1}{80}$ grain, and in the next week she had none, but in the following two. The dose was then increased to $\frac{1}{50}$ grain, and no other fit occurred during the next seven weeks, when she left the hospital. She was heard of four months later as having continued the medicine and having been perfectly free from attacks. The dose was then reduced to $\frac{1}{100}$ of a grain, and three months later, when last heard of, she was perfectly well.

I have not obtained a similar result in any similar case. But there is one class of patients in which nitroglycerine is often of distinct service. They are cases of minor attacks in young children, after actual infancy, between three and twelve years of age, in which there is sudden brief unconsciousness, often suggesting a simple momentary faint, in which pallor of the face quickly develops, although not often present at the actual onset. The following instances illustrate the value of nitroglycerine in such cases, combined with strychnia, which I have not found effective alone.

A boy, aged 7, had had one fit in infancy during teething. There had been no recognised recurrence until a month before he was seen, and during that time he had had five attacks of momentary loss of consciousness. On $\frac{1}{300}$ of a grain of nitroglycerine ($\frac{1}{3}$ of a drop of Liq. trinitrini) and some strychnia he had no other attacks for six months, when two occurred of similar character, but preceded by a momentary pain in the stomach. For two years he had no other attack. Then the medicine was left off, and in six months he had another attack of the same kind, followed by sleep. The medicine was resumed, and continued for three years, then gradually reduced and omitted; he has now passed five years without any recurrence.

A girl, aged 9, began to suffer at 8 from momentary drooping forwards of the head, and afterwards, with it, cessation of speech for a moment; after a time there was also a fall. In addition to these attacks in the day, she became liable to peculiar seizures in the night, described as choking in the throat, with spasmodic movement in the eyes and jaws, followed by loss of speech for five minutes. The attacks had occurred as frequently as twenty-three in the day. (I was consulted in this case by letter from South America, the doctor and father sending me the particulars). Twenty grains of bromide a day had been given without any influence on the attacks. I recommended half a minim of solution of nitroglycerine and three minims of Liq. strychniæ, the former to be increased gradually to one minim. But it was not increased. Until the arrival of the advice the attacks went on with the same frequency and character. The day after the medicine was commenced the attacks ceased, except a few gasping attacks in the night. In a few weeks these had also ceased, and she remained perfectly free. She came to England six months later, and was perfectly well, although the medicine had been discontinued on the voyage.

ZINC.

Zinc unquestionably deserves some of the repute which it has enjoyed for more than a hundred years, as a remedy for epilepsy. Its influence is rendered intelligible by the fact that, given to animals, it has been found, in large doses, to lessen reflex action, and also is said to influence the functions of the cerebral hemispheres. In the pre-bromide days, it was one of the drugs to which most value was ascribed, and it was largely used and praised by Herpin. Of late years it has been comparatively neglected, certainly more than it deserves. It very commonly lessens the frequency of attacks, but in most of the cases bromide has a far more powerful effect. In a few cases in which bromide fails to do good, zinc is of distinct service, and attacks may even cease entirely under its influence. It is certainly most often useful in cases in which the attacks are slight or moderate in degree, very seldom in those with severe convulsions.

I have tried many salts of zinc, the oxide, lactate (recommended by Herpin), citrate, sulphate, and bromide. The latter can only be given in doses of two or three grains, on account of its irritant effect. On the whole, I think that the oxide is to be preferred. The dose which the stomach can tolerate varies much in different persons. In some, 3 grains well diluted, after meals, causes nausea; in others, 7 or 10 grains can be borne. A small quantity ($\frac{1}{10}$ gr). of hydrochlorate of cocaine often lessens the gastric irritation; but it is far better to give the zinc in the form of a pill. This may be taken at the same time as a dose of bromide, if it is desired to give the two together. It is no doubt converted in the stomach into chloride of zinc, but so slowly as to cause no irritation.

The following cases are mentioned as evidence of the definite influence zinc often exerts. The difficulties in discerning the permanence of its effect are the same as in the case of bromide and of all other agents.*

A man, aged 28, whose mother's sister was insane, had his first fit, without known cause, at 27. He had been occasionally intemperate. The attacks occurred in batches of seven or eight every six weeks, always when he was awake. Each was preceded by a visual aura and a vague mental sensation. They were severe, with tongue biting. On five grains of oxide of zinc twice a day, he passed five months without an attack, and then ceased attendance.

A girl, aged 8, with inheritance (paternal grandfather and great-aunt), had had fits for a year, at intervals of a few days to two months. The warning was sudden headache, and she would fall unconscious, sometimes bite her tongue, and often pass into a post-epileptic hysteroid state. After four months of bromide treatment she still had two fits a week. Three grains of oxide of zinc were ordered, and in two months the fits had ceased, and she continued free for six months. She then had an attack, but no other for three months more.

A man, aged 26, had suffered for eleven years from fits, which occurred once a fortnight, usually in the morning; they were unattended by warning, but he would fall suddenly and then pass into a condition of hysteroid convulsion. In some attacks he bit his tongue, and the hysteroid convulsion doubtless succeeded a slight epileptic fit. Five grains of oxide of zinc was given twice a day, and for two months he had no attack. In the next two months he had two fits, so the dose was raised to ten grains, and he had no other attack during three months longer that he remained under treatment.

Occasionally the addition of zinc to bromide distinctly increases its influence over the attacks.

A dull, stupid boy, whose sister was epileptic, began to suffer from fits at eleven months, "from teething," and they were occurring once a fortnight when he came under treatment at fourteen. The warning was "giddiness." They occurred both by night and day. On bromide of ammonium (℈j three times a day), the fits ceased for several months, and the lad discontinued attendance, but returned in consequence of a recurrence. On the same treatment, and on bromide and belladonna, the attacks continued very frequent, and although they were lessened by bromide and digitalis, they did not cease until oxide of zinc was substituted for the digitalis, the dose of bromide remaining the same. The attacks then at once ceased, and he had no attack for fourteen months.

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* The opportunities for a valid inference regarding permanence of effect seldom present themselves to the physician. They are chiefly met with by those who are engaged in general or family practice.

I could add many other cases in which attacks ceased under zinc alone, or in combination with bromide; and others in which zinc arrested when bromide did not; but the results were only discerned during the few months the patient remained in the hospital, too short a time to give the facts definite weight. When zinc seems to do good and is well borne by the stomach, the dose should be increased to 8 or 10 grains three times a day.

OTHER AGENTS.

Iron.—Certain distinguished authorities (as Brown-Séquard and Hughlings Jackson) have discountenanced the administration of iron to epileptics, believing that, while it improves the health of epileptics, it increases the frequency and severity of the fits. I believe that this conclusion is only partially correct. I have given iron to several hundred epileptics, for both long and short periods, and have carefully watched its effect. Its influence certainly varies in different cases, and may be thus summarised. In rare cases it does increase the frequency of the attacks; in the majority of cases it may be taken without any ill effect on the attacks, and often with all the benefit to the general health which attends its use in other cases in which it is indicated. In some patients its influence on the attacks is distinctly beneficial. Attacks which continue on bromide alone may cease when iron is added to it, and a few patients are better on iron only than on bromide only. I have, indeed, met with cases in which attacks which continued on bromide ceased altogether when iron was substituted.

The cases in which the attacks are distinctly aggravated by the administration of iron are so uncommon that no hesitation on this ground need ever be felt in giving iron to an epileptic in addition to the bromide or other drug which he may be taking. If it is *substituted* for bromide, the effect of the cessation of the bromide must not be ascribed to the influence of the iron.

The beneficial influence of iron in epilepsy is not limited to its hæmatinic effect. It appears to do good by a specific action on the nerve-centres, similar to that which is produced by some other metals. Meyer and Williams* found that iron has such an action. They injected tartrate of soda and iron into the blood of various animals, and found that it caused motor paralysis of central origin, the excitability of the muscles and peripheral nerves remaining intact. Its influence in pure epilepsy is illustrated by the following case.†

A single man, aged 23, had his first fit five months before he came under my care. No cause could be assigned for it. There was no history of inherited or acquired syphilis. The interval was rather less than a week. They occurred usually at night. The convulsion was severe, his tongue being often bitten. He was ordered tinct. ferri perchlor. ♏x three times daily. During the next month he had only one slight attack; during the following month he had no fit; during the third month he had only one fit; during the succeeding three months he had not a single attack, and he then ceased attendance. During the whole of this time he took the iron only. After ceasing to take it he remained free from fits for four months. The fits then returned, and three months later he came again to the hospital, having the fits 'as bad as ever.' He was again put on the perchloride of iron, and the fits at once ceased; he attended the hospital for three months more, and during that time had not a single attack.

Mistletoe.—This old remedy for epilepsy, praised by Paracelsus and by a considerable number of writers since his time, especially by Colbatch (1723) and Fraser (1806), I have tried in several cases, but with no beneficial result.

Turpentine was originally recommended by Pritchard and Foville, and has been praised by Watson and Radcliffe. I have, however, seen no good result from its administration in epilepsy, although in hysteria it is sometimes distinctly useful.

Cocculus Indicus, Picrotoxine.—The use of cocculus indicus in epilepsy was introduced by Dujardin-Beaumetz. The alkaloid, picrotoxine, has been chiefly employed. Many years ago I had an opportunity of watching an extensive series of observations on the effect of hypodermic injections of the alkaloid, undertaken by the late Dr Ramskill. His experience of its effect on epilepsy when given through the skin, agrees with my own experience of its administration by the mouth, that it exerts no appreciable influence on the attacks. A remarkable fact was, however, ascertained by Dr Ramskill, —viz. that picrotoxine in larger doses of from fifteen to eighteen milligrammes will almost invariably produce a fit in twenty or thirty minutes.

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* 'Archiv für Exp. Path.,' Bd. xiii, p. 70.

† In the first edition many cases were narrated to illustrate the impunity and advantage with which iron may be combined with bromide. The objection to its use has, however, largely lessened during the last twenty years, and it is not worth while to repeat them.

We cannot assume with certainty that picrotoxine and cocculus indicus are identical in their effect. The following case is the only one in which cocculus indicus seemed effective:

A girl, aged 16, began to suffer from fits, without known cause, a year before she came under treatment. She had only severe attacks once a month, always in the early morning. Each was preceded by a sensation in the hypogastric region, felt on waking, and this sensation "worked up to the heart," and then she lost consciousness. The fit was epileptic, lasting two or three minutes. Five minims of tincture of cocculus indicus three times a day were ordered. During the first month she had no fit; during the second, one only; during the third, one, but that occurred after she had omitted the medicine for a few days. The dose was then increased to ten minims. During the next four months she had not a single attack, and she then ceased attendance. Six months later there had been no recurrence.

Chloral hydrate I have not found useful in ordinary cases of epilepsy, alone or in combination with bromide. From *nitrate of silver* and *sulphate of copper*, remedies which in the past enjoyed a high repute, I have seen, in the few cases in which I have employed them, no resulting benefit.* Among other drugs which I have tried and found useless are *benzoate of soda*, bromide of aluminium, *Piscidia erythrina*, *Codeia*, *Calabar bean*, *Ergot*, *Sclerotic acid*, and *nitrite of amyl* (except for the arrest of attack).

Among agents that have been tried by others and found without effect are, *Curara*, *Hydrastin*, *Chinolin*, *Resorcin*, *Antipyrine* (Lemoine), *Acetanilide* (*antifebrine*, Salm), *Amy-*

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* Many years ago, among the out-patients at the hospital was a man who had been deeply stained by nitrate of silver, and was still suffering from the disease.

lenhydrate (Wildersmuth). The last quickly loses any influence it possesses, but has been thought useful in very frequent attacks. *Osmic acid* in a dose of $\frac{1}{10}$ grain in a pill has been thought to exert a trifling influence, but by others has been found useless. It has been said to lessen the excitability of the motor centres of the cortex. *Thyroidin* may be added to the long list of agents which have not been found of service.

STATUS EPILEPTICUS.

When severe attacks succeed each other so frequently that there is no return of consciousness in the intervals, or only an imperfect return, the condition is one of "status epilepticus."* If it continues, it quickly becomes one of obtrusive danger, and taxes to the utmost the efforts of therapeutics. The state has been already described (p. 207).

When, as is often the case, it follows the omission of bromide, the administration of this in full doses is important. Thirty grains should be given by the mouth, or 60 by the rectum, every four or six hours. It is, however, more effective in slight than in very severe attacks of the status. It may be stopped if no influence is produced in the course of twelve or fourteen hours. If the condition comes on in a patient who has been steadily taking bromide, little benefit is to be expected from increased doses of this. In most cases, moreover, other agents seem to have more influence; they may be combined at first with bromide, which may be omitted if it seems ineffectual or harmful. The most useful are the following.

Chloral hydrate, especially in combination with bromide It may be given in doses of 20 or 30 grains by the mouth, 40 or 60 by the rectum. The inhalation of chloroform often causes an arrest of the attacks, and, although this is usually temporary, it is useful as affording time for agents given by the mouth to exert their influence. Nitrite of amyl may arrest individual attacks, but has no permanent influence. An agent of special value is the hypodermic injection of hydrobromate of hyoscine; $\frac{1}{150}$ or $\frac{1}{100}$, or even $\frac{1}{50}$ of a grain may be given, and repeated in four or six hours. It should be lessened or omitted as soon as its influence is perceptible. A diminution in the attacks which it has effected is often increased and maintained by bromide. Morphia has been injected with great success in some cases, but only in doses which are fraught with danger, $\frac{1}{4}$ or $\frac{1}{3}$ gr. Those which may be safely employed ($\frac{1}{12}$ and $\frac{1}{10}$) are seldom effective, although they may sometimes deserve a trial if other measures fail; the small doses may be given every two hours and the effect watched. But it is not likely that morphia would be successful when hyoscine is not. Any serious sign of cardiac depression is best met by the hypodermic injection of strychnia, $\frac{1}{50}$ or $\frac{1}{30}$ of a grain.

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* We have no English equivalent for the Latin term or its French rendering, "l'etat du mal epileptique," and it is not easy to invent one. A literal translation, "epileptic state," is devoid of the pronounced significance which should be conveyed. We may, however, anglicise the word "status" in this application since it is unappropriated in medicine.

In all cases of the status, the maintenance of the strength of the patient is most important. It is impossible to forecast the duration of the demand on the strength of the patient, which should be kept up from the first as much as possible. Food that is readily swallowed and easily digested should be given by the mouth, by nasal tube, or by the rectum. Stimulants should be regulated by the state of the pulse.

The application of ice to the head may be tried, but has not often been effective. Dr Mott has called attention to the fact that destructive changes in the nerve-cells of the cortex occur only in cases of the status in which the temperature rises to a hyperpyrexical degree, which may be regarded as exceeding 103° or 104°. He urges, therefore, the importance of reducing the temperature of the body in such cases by cold baths. The advice, although reasonable, has not yet received much practical confirmation; but in one such case, a child of six, with a temperature of 104·5°, the convulsions were at once arrested by immersion in cold water.* Without hyperpyrexia, cold baths seem to do more harm than good.

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* Case related to me by Dr. Collier, Pathologist to the Queen Square Hospital.

TREATMENT OF ATTACKS.

Arrest.—In the case of fits which are ushered in by a deliberate warning, the attacks may sometimes be cut short at the onset. The means by which this may be done have been already considered (p. 111). The attempt is most frequently successful in cases of convulsions beginning unilaterally in the hand or the foot, in which a ligature around the limb, or the forcible extension of the contracting muscles, is sometimes effective. The most useful way of employing the ligature is for the patient to fasten a piece of tape round the arm so that it can readily be tightened. If the tape is doubled, passed round the limb, with the ends through the loop formed by the doubled part, the extremities may be brought down to the lower part of the sleeve, so as to be accessible and readily pulled tight, as soon as the warning is felt.

The method is occasionally efficacious, and may even produce a more permanent effect. I have already (p. 114) mentioned the case of a patient whose commencing attack stopped spontaneously at the spot at which the ligature had previously been applied. A similar case was recorded in the last century by Lysons.*

It has been proposed by Brown-Séquard and by Buzzard to produce a more permanent effect, in the cases in which the ligature is effective, by applying a circular blister around the limb.† In some cases this method is effective. In one patient, described by Buzzard,‡ it led to a transfer of the commencement of the fits from one arm to the other, and some years later, the patient having died of phthisis, I found that there existed a small tumour in the white substance above one lateral ventricle.

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* 'Pract. Essays upon Intermitting Fevers,' 1772.

† The method is not entirely modern. Alexander Trallianus, who lived soon after Galen, describes a case in which the attacks commenced by a sensation beginning on the back of the foot and ascending to the head. The fits were arrested by creating an issue on the part from which the attacks seemed to start. The treatment by local blisters was employed also by physicians in the seventeenth and eighteenth centuries.

‡ 'Practitioner,' Oct., 1868.

The effect of the ligature and blister alike must be ascribed to the stimulation of the sensory structures in the brain, and an inhibitory influence exerted on the motor structures. Cases are met with in which the effect seems to cause wider disturbance, and was attended by so much giddiness and other distressing feelings, that the patients declared that the arrested fit was worse than the full attack.

Other means of increasing the central resistance by peripheral impressions, especially by daily faradisation of the limb with a wire brush, and by circular sinapisms, I have not found effective.

Attacks which begin by other warnings are sometimes arrested by other forms of peripheral sensory impression, by a strong gustatory sensation, as by chewing a piece of ginger; by a strong olfactory impression, as by the application of ammonia to the nostrils, or by swallowing a handful of common salt (Nothnagel). More uniformly successful, however, is the inhalation of nitrite of amyl. If it produces flushing of the face before the patient loses consciousness, the attack is usually cut short. The cases in which I have found it most frequently successful have been those with a deliberate olfactory aura. Why this should be is difficult to explain, since it is scarcely conceivable that the effect of amyl is produced through the olfactory nerve. A sudden increase in the blood-supply to the brain may change the form of cerebral action, whatever be its original cause, when it is sufficiently deliberate. The patient whose deliberate warning is described on p. 78 would not, on any account, have been without his bottle of amyl, and it was equally useful in the case of another patient with an olfactory warning. An incident which once occurred to this patient may be mentioned as illustrating a possible inconvenience of the drug. One day, when out, he felt an attack coming on, and smelt the amyl, but too late to arrest the fit. He fell unconscious, spilling the contents of the bottle in his fall. A person a few yards away, who saw him apply the bottle to his mouth and at once fall down insensible, rushed to him, and, seeing the empty bottle labelled 'Poison,' sought the nearest policeman, and asked him to go to a man who had just committed suicide. When the policeman arrived at the spot, he found, to his surprise, the supposed suicide preparing to walk away. Such an accident may be prevented by the use of the glass capsules of nitrite of amyl now to be obtained.

Treatment during an Attack.—Little treatment is needed during the attacks of epilepsy. In patients who bite the tongue, a cork, a small piece of indiarubber, or any suitable object that is at hand, should be forced between the teeth, and thus the tongue-biting may often be prevented. The patient should be laid down; whether with the head low or high matters little.

It is necessary to loosen the clothes about the neck. If tight, when the neck becomes turgid and swollen, the resistance to the return of blood from the head is increased, and extravasations into the skin or conjunctivæ are more probable.

Little can be done to arrest the developed attack. Only in the early stage is an attempt to cut it short by any method successful. The sleep which in many cases succeeds a fit should not be prevented. There is more headache if the patient is kept awake than if permitted to sleep. The administration of a small quantity of alcohol after an attack does no harm, but it is rarely needed or beneficial. The weakness and prostration pass away in a little time without its aid.

It is important to note the movements of the chest and throat that indicate impending vomiting. The patient should then always be placed on the side, lest substances brought up from the stomach fall back into the larynx—an occasional, though very rare, cause of death. Only one instance has come within my personal knowledge. A more frequent source of peril is the tendency to turn over on the face, chiefly dangerous in nocturnal fits. Inquiry should be made for it, and the friends warned of the risk. (See p. 125.)

Hysteroid Attacks.—In the management of a severe attack of this kind, the most important point is abstinence from that to which there is an invariable tendency—restraint. All restraint intensifies the struggling movements which are the object of the restraint. The more force is employed the more is needed. The patient should be allowed free play, and it is surprising how few even bruises will result. Such attacks, when post-epileptic, usually cease in a few minutes. If they do not, it may be desirable to cut them short. This can often be effected by a strong sensory impression of any kind, especially one that is unfamiliar, such as a strong induced current applied to one of the limbs. A magneto-electric apparatus answers well.* Affusion with water is often employed, but more than one jugful is generally needed. A small quantity will suffice, if poured into the mouth of the patient. It acts then by the same mechanism as the well-known expedient, recommended by the late Dr Hare, of closing the mouth and nose with a towel until the patient is on the point of asphyxia, when all convulsion ceases. The chief disadvantage of this method is the impression it conveys to the friends. Unquestionably the most effective measure of arresting severe attacks, which would otherwise go on for an hour or two, is the injection of apomorphia. One sixth of a grain may be injected, at any practicable moment during the violent contortions. It was found at the hospital that in two minutes all spasm ceased, and the patient began to look uncomfortable. In three minutes she got up and walked to the nearest sink; and in four minutes vomited copiously; and the after effect on the attacks was usually enduring. These were cases of pure hysteria, and not of post-epileptic hysteroid convulsion, in which the measure is hardly ever needed. These are always much slighter, and seldom need any special measures to cut them short,—at least, not more than a wet towel or a minute's faradism.

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* I have heard of an old country surgeon who employed, with success, as a method of arrest in all hysterical fits, a vigorous tug at the pubic hair. Its utility is intelligible.

GENERAL REGIMEN AND MANAGEMENT.

Diet.—It has been recommended, on theoretical grounds, that the diet of epileptics should contain no animal food. In a series of observations which I have made by keeping a series of patients under unaltered medicinal treatment and general conditions, for alternate periods on a diet with and without animal food, I could observe no difference in the attacks, except that in one patient they were slightly more frequent in the periods when animal food was excluded, and in another they became much more severe.

There is, however, reason to think that many patients are better if meat is taken only once a day, and that beef should be taken sparingly. I have met with one case in which beef invariably induced an attack, although mutton could be taken with impunity. There was no doubt of the relation, and that which has occasionally so pronounced an effect, is likely often to have some unfavourable influence.

But the question of diet depends very much on the amount of food taken. In weakly, ill-nourished patients, with habitual anorexia, the kind of food matters little so long as it is digestible. On the other hand, many epileptics have a tendency to eat too much and too fast. Each should be prevented as far as possible.

In all cases it is important to avoid indigestible food, tough or salt meat, hard bacon, raw apples, raw vegetables, carrots and parsnips, and especially the skins of fruit, whether fresh or dried. Even currants should be avoided, for their skins pass unchanged through the intestines. Nuts of all kinds should, of course, be absolutely prohibited.

Stimulants should be taken sparingly by epileptics. In young persons who have not been accustomed to stimulants, alcohol is better avoided altogether.

In all cases it is most desirable that regular action of the bowels should be secured. Nothing conduces to the occurrence of attacks more than constipation and gastric disturbance.

EDUCATION AND OCCUPATION.

The arrangements of the elements of the life of the subject of epilepsy is often a problem of much practical difficulty. Many points have to be taken into consideration and to receive their proper weight. Age, intellectual development, mental tendencies and inclinations, have to be considered in relation to the external conditions of life.

At all ages and in all states it is important that excitement and mental strain should be avoided. Mental work, such as constitutes exercise without strain, is seldom undesirable. In most cases it is as beneficial to the mind as physical exercise to the body. There is too great and too general a tendency to consider that the education of children who become subject to fits should cease, as long as the attacks continue. But moderate and careful mental training rarely seems harmful. Prejudicial effects are usually to be traced rather to the excitement of the playground, or the mental discomfort often inseparable from contact with other children, than from the work itself. The ultimate course of epilepsy can never be foreseen. However great the hope, or good the prospect, that the disease may be arrested, it is important to realise that its persistence is possible, and that education should not be neglected, but should, in degree and character, fit the patient for such kind of work in life as his position permits. There are, of course, many patients for whom any education is useless, but there are also many who are handicapped in after life by unnecessary neglect of earlier mental training. Any degree of mental work which induces the attacks should, of course, be reduced; but in all cases, except those who must be regarded as imbecile, there is some form and degree of exercise of the mind which can be borne, and is beneficial. What this is varies in every case, and must be decided after careful observation, which need only be guided by the practical wisdom which is termed "common sense."

Similar principles must be applied to the question, often most perplexing, What should be the occupation of a lad who is, or has recently been, liable to epileptic attacks? Necessarily much depends on circumstances; much also on the individual. Some are hopelessly unfitted for any occupation. Many, indeed most, are capable of such work as may "earn a living;" a few of more.

In every case of epilepsy, however great may be the hope that the attacks of youth will cease, it is never wise to allow this hope to influence the decision as to the nature of the occupation. If one is chosen which can be followed, even if attacks recur, nothing is sacrificed; if, on the other hand, the anticipation that an arrest of attacks will persist, or their arrest may be looked for in the future,—if this leads to the choice of an occupation which could not be pursued if attacks recurred, all the time devoted to its acquisition is lost if the malady recurs. On the other hand, work during which an occasional fit would entail inconvenience, but no danger, is devoid of this risk. The choice of safe callings is limited, almost, to those which are sedentary. Any occupation which takes the sufferer on scaffolding, up rigging, among machinery, into any situation in which a fall, even without unconsciousness, might involve danger to life, is unsuitable.

The friends of patients are often concerned regarding the advantage of an out-door occupation. Such a life does promote general health, but I have not met with any definite evidence that it exerts an influence on the liability to attacks. Unless the choice is very free, it is wiser to select the best of those that involve no danger. Farming and land-surveying are the most suitable of those which involve fresh air, but they are not often within reach.

MARRIAGE.

The question of marriage* presents itself in two aspects, as regards the individual, and as regards the possible offspring.

(1) As regards the disease, marriage in itself has little in-

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* An interesting account of the medical, popular, and legal opinions on the relation of marriage and epilepsy has been given by Echeverria, "Marriage and Hereditariness of Epileptics," 'Journal of Mental Science,' October, 1880.

fluence. The ancient opinion that continence may cause epilepsy, or incontinence cure it, is probably now held by none, and is certainly as opposed to facts as it is unfortunate in its practical effects. Attacks which have resisted treatment before marriage usually persist afterwards without any considerable change. There is no evidence to show that marital relations, in moderation, have any influence on the disease. In women (as well as in men) the attacks continue, as a rule, without material change, unless pregnancy occurs. The influence of this has been already considered (p. 211). There is seldom any enduring effect. Nor does epilepsy seem to increase the danger of parturition or the puerperal state.

(2) As regards the offspring, however, the question of the likelihood of the transmission of the disease depends on the conditions of its origin.

(*a*) We must consider apart the large number of cases of what has been termed "organic epilepsy," in which the character of fits, and of the initial attacks, and also the conditions under which these occurred, indicate that they depend on a local lesion on the surface of the brain. (See p. 154). In such cases there is no reason to fear the transmission of the disease, unless there is also evidence of heredity. All the facts at our disposal confirm the conclusion that acquired conditions, however severe may be the functional disturbance, and however early in life may be their origin, are not transmitted. The subject has given rise to a vast amount of controversy, but the facts justify the inference that has been stated.

(*b*) The danger of transmission occurs in the cases in which there is evidence of inheritance, in which epilepsy or insanity has occurred in direct ancestors or in collaterals, in past or present generations. That the actual facts far transcend those that can be ascertained, is certain, and has been before emphasised. (See p. 14.) The occurrence of such diseases is either unknown or concealed, and the knowledge of that which is thought to be a stigma, has often ceased with the death of those who possessed it. Hence, even slight evidence of antecedent or collateral disease, of fits, repeated faints, insanity, or suicide, must be regarded as probably implying a morbid disposition greater than is manifest. Whenever evidence of inheritance can be discerned, the danger of transmission is definite, and cannot be ignored. The amount of risk is only roughly proportioned to the extent of traceable antecedent disease. Exceptions are frequent in the relation between the two. Consideration of the facts, however, suggests that, if there are six children, the chances are against the escape of all from epilepsy, insanity, or imbecility. But the chances are considerably against the affection of any single child. In some cases more than one suffers; in many, all escape. As in all questions of probability, the rules which are true of a number taken together, often fail in individual instances. No precise forecast can therefore ever be given.

The feelings which induce marriage are usually too strong to be amenable to the doctrine of probabilities. It would, however, certainly be for the welfare of the community if members of families with clear inheritance abstained from the risk of transmitting disease. It is difficult to advise those who seek guidance on the subject, especially as it is chiefly desired by those who are conscientious, often to a painful degree. It is best to ascertain carefully the facts, and state candidly the prospects, and leave those concerned to decide. When, however, there is distinct heredity on the side of both intending wife and husband, a physician's duty should, I think, carry him further, and he should distinctly deprecate the union. It must be realised that, apart from the question of the general health of the community, which the individual can lightly toss aside, personal happiness is often involved. It is distressing to witness the anxiety to parents, and their often lasting sorrow, when a child becomes the subject of epilepsy or insanity. The sorrow sometimes spares the immediate generation, and falls on the next.

(*c*) But there is a large class of cases in which it is very difficult to give an opinion. They are the cases in which the disease has all the features of idiopathic epilepsy, and commences without an adequate cause, but in which no inheritance can be traced. In some cases the disease has been preceded by infantile convulsions, such as are due to retarded development. If the evidence of such retardation is considerable, and the infantile convulsions have the characters which indicate their dependence on this cause (see p. 21), their influence on the brain seems sometimes to induce epilepsy, which may be regarded as purely acquired, without danger of transmission. But many cases of this class must be looked on with suspicion, especially those in which the infantile convulsions seem inadequate, without the concurrence of a congenital disposition. In some of these, we cannot doubt, there is unknown heredity. Antecedent cases are often denied, and subsequently ascertained by more careful inquiry. Other cases, however,—how many we cannot tell,—involve risk of transmission because the disease is due to a tendency which is congenital, though not inherited. All hereditable diseases and tendencies seem sometimes to arise *de novo*, in the elements from which the embryo develops. Sometimes more than one member of a family may suffer, as in the cases of congenital blindness and of epilepsy mentioned at p. 12, although no antecedent disease can be ascertained. But we meet with cases apparently isolated (as far as the most far-reaching scrutiny can ascertain) of certainly congenital diseases in pseudo-hypertrophic paralysis, and in the so-called "hereditary ataxy." In such cases we cannot feel sure that the embryonal defect, which is the source of the disease, may not be transmitted. Congenital conditions and tendencies, congenital in the strictest sense, may exist without inheritance, and may then probably be sometimes transmitted. Such conditions seem to be acquired, but are not, in the ordinary sense of the word.

It is thus very difficult to give advice to cases of this class. As just stated, when there have been severe "teething" convulsions, with characteristic signs of rickets, no evidence even of allied neuroses, and the fits have apparently grown out of the infantile attacks, the case may be regarded with some confidence, as belonging to the class of acquired epilepsy in which marriage need not be prevented by fear of transmission. With regard to the cases without heredity which have no such obtrusive infantile causation, but begin later in life, with no adequate cause, and present all the features of the idiopathic disease, it is impossible to form or express a reassuring opinion. We cannot exclude their dependence on a morbid tendency inherent in the elements from which the embryo proceeds, but we cannot affirm it. Such cases, regarding the risk of transmission, are among those of which it is only possible with truth to answer, "I cannot say."

SURGICAL MEASURES.*

Counter-irritation, by applications to the skin or the scalp or neck, has been frequently employed, the usual method being a seton in the neck. That this occasionally does good is testified by strong, though ancient, evidence. An extensive accidental burn sometimes produces a similar effect. But in most cases the relief seems to have been temporary. In the few cases in which I have tried a seton, no effect on the attacks could be discerned.

Ligature of the Vertebral Arteries, both arteries being tied at the same time, was a procedure adopted, and for a time recommended, by Dr Alexander, of Liverpool. It does not now need more than a mention. The unsatisfactory results and the risks of the operation have led to its abandonment, even by its proposer.

Resection of the Cervical Sympathetic, the nerve being simultaneously excised on the two sides, has been employed by some French and Roumanian surgeons. It rests on no valid theory, and the recorded results are not of a character to make it necessary to describe them or to discuss the proceeding.†

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* The importance of the operative arrest of any traumatic irritation in the cerebro-spinal system, distinctly exciting attacks, has been already mentioned (p. 258).

† A careful consideration of the subject is given by Cullère, 'Ann. Méd.-Psycholog.,' 1898. The operation is an old one, revived by Jonaesco, and an account of a discussion on it will be found in the 'Gaz. des Hôp.,' 1898.

TREPHINING.

Trephining is a very old remedy for epilepsy, which has come again into great prominence in consequence of the more exact localisation of diseases of the brain. But cases of epilepsy need to be considered in several distinct classes in relation to the operation. (1) Idiopathic epilepsy; (2) organic epilepsy without, and (3) with, indications of cranial injury.

(1) *Idiopathic Epilepsy*, in which there is no reason to suspect local disease of the brain, in which the convulsions have always been general, and in which minor attacks often occur. Simple trephining, without the removal of any part of the cortex, has been employed in these cases, chiefly in America, and especially recently by Kocher in Berne. When any theory has underlain it, it has been a vague idea of increased intra-cranial tension, which might be relieved by the operation.* In very few cases has the operation had any influence on the disease, when the cases have been observed long enough to enable a just conclusion to be formed. It is consistent with what we know of cerebral function to assume that so disturbing a procedure should cause a temporary arrest of the attacks, and that now and then the influence should be prolonged, by virtue of an influence analogous to that of a counter-irritant. When the cases which have been insufficiently observed are put aside, and the character of the disease is considered, the operation cannot be regarded as justifiable in any case of idiopathic epilepsy. Even in cases associated with idiocy, in which increased tension is supposed to have resulted from premature closure of the sutures, the effects of the operation have been generally unsatisfactory.

(2) *Organic Epilepsy without Signs of Injury to the Skull.*—These are cases in which the character of the early convulsions, and of the slighter recurring fits, indicate that the discharges proceed from a definite region of the cortex,

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* For a conclusive disproof of a causal relation between attacks and intracranial tension see Nauratzki and Arndt, 'Berlin. klin. Wochenschr.,' 1898, No. 30.

which raises a strong presumption of organic damage. In some there is a history of a fall or blow on the head in early life, but with no external evidence of injury. In many cases of this class the severity and unilateral character of the initial convulsions, and the hemiplegia which often attends them, proves local disease, cortical encephalitis, or thrombosis in a branch of a vein. These indications have been already discussed. All cases of this class agree in presenting evidence of an old, stationary lesion, in or near one of the motor centres. Cases in which there is reason to suspect progressive disease, such as a tumour, are, of course, outside the present subject.

In such cases the question always arises of the desirability of trephining, and usually of opening the dura mater, and removing any distinctly diseased part of the cortex. Simple trephining cannot be expected to have any influence, except in the rare cases in which there is depressed bone without any evidence of external damage.

The removal of a portion of the motor cortex is almost certain to be followed by weakness in the related region of the opposite side, and this, in the case of the arm, is often permanent. In these cases severe convulsions usually affect the second side; their frequency, and the length of time during which such general convulsions have occurred, and the readiness with which convulsions of moderate severity become general, are important indications of the general instability which has probably been induced by the discharges starting from the diseased region. This indication, in proportion to its degree, lessens the probability of the attacks being arrested by the removal of the source of the original discharge.

An unfavourable result is also suggested by a feature, which many of these cases of organic epilepsy present when the attacks have occurred for many years, and especially when there have been frequent general convulsions—the occurrence of minor attacks quite similar to those which characterise idiopathic epilepsy. These also are evidence of an acquired state of the brain, widely distributed, indisposing to the cessation of attacks.

It has been advised that in all cases in which there is evidence of such secondary instability, bromide should be given for at least six months after the operation.

The cases in which trephining and removal of any cortical disease may reasonably be adopted, are those in which there are no such minor attacks, in which only the most severe attacks become general, and the second side is then affected deliberately after the first, and in which there are many local attacks, involving only the part in which the convulsions begin. The more frequent and dominant these are, the greater is the indication for an operation. But in all cases the certainty of some loss of function from an operation on the cortex has to be set against the probability of good, and the latter should be definite to justify the procedure.

It should be remarked that not all cases in which epilepsy follows a fall on the head, are cases of such local lesion as justifies an operation. It is not uncommon to meet with patients in whom attacks, which have always been general, and have presented the features of idiopathic epilepsy, followed a fall or blow on the head. It seems as if the disease may result from the general impairment of nutrition and function which results from a concussion of the brain. Without local signs, indicative of focal disease, traumatic causation does not justify operation.

On the whole, however, an operation is more often justified by success in the case of organic epilepsy, with convulsion of local onset, following a blow, than in cases in which the causal lesion is the result of disease.

It should be remembered, however, that local onset is not an unerring indication of an organic lesion, although it must be due to local instability. It is met with in rare cases in which gradual and spontaneous onset, with hereditary disposition, make it probable that the disease is idiopathic. The conclusion is sometimes confirmed by a variation of the local onset from one side to the other (see p. 57).

(3) *Cranial Injury.*—The third class of cases, in which the question of trephining presents itself with special force, comprises those in which there are external signs of damage to the bone of the skull, or persistent tenderness at the seat

of the blow, in the neighbourhood of the motor centres, and in which the fits begin in the limb corresponding to the centre beneath or near the seat of injury. The operation in such a case is almost a surgical compulsion. It is said that the prospect of success is not lessened by the interval between the injury and the first fit. Even in these cases, however, the considerations mentioned in connection with simple organic epilepsy must be taken into account. The readiness with which the convulsions become general, and the length of time such generalisation has occurred, influence the prospect of success. They should not, however, prevent an operation when the external signs of damage are distinct. Whether or not depressed bone is found, or disease outside or of the dura mater, the importance of opening this is generally recognised on account of the frequency with which a bruise of the cortex is found. With antiseptic measures, little additional risk is entailed.

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